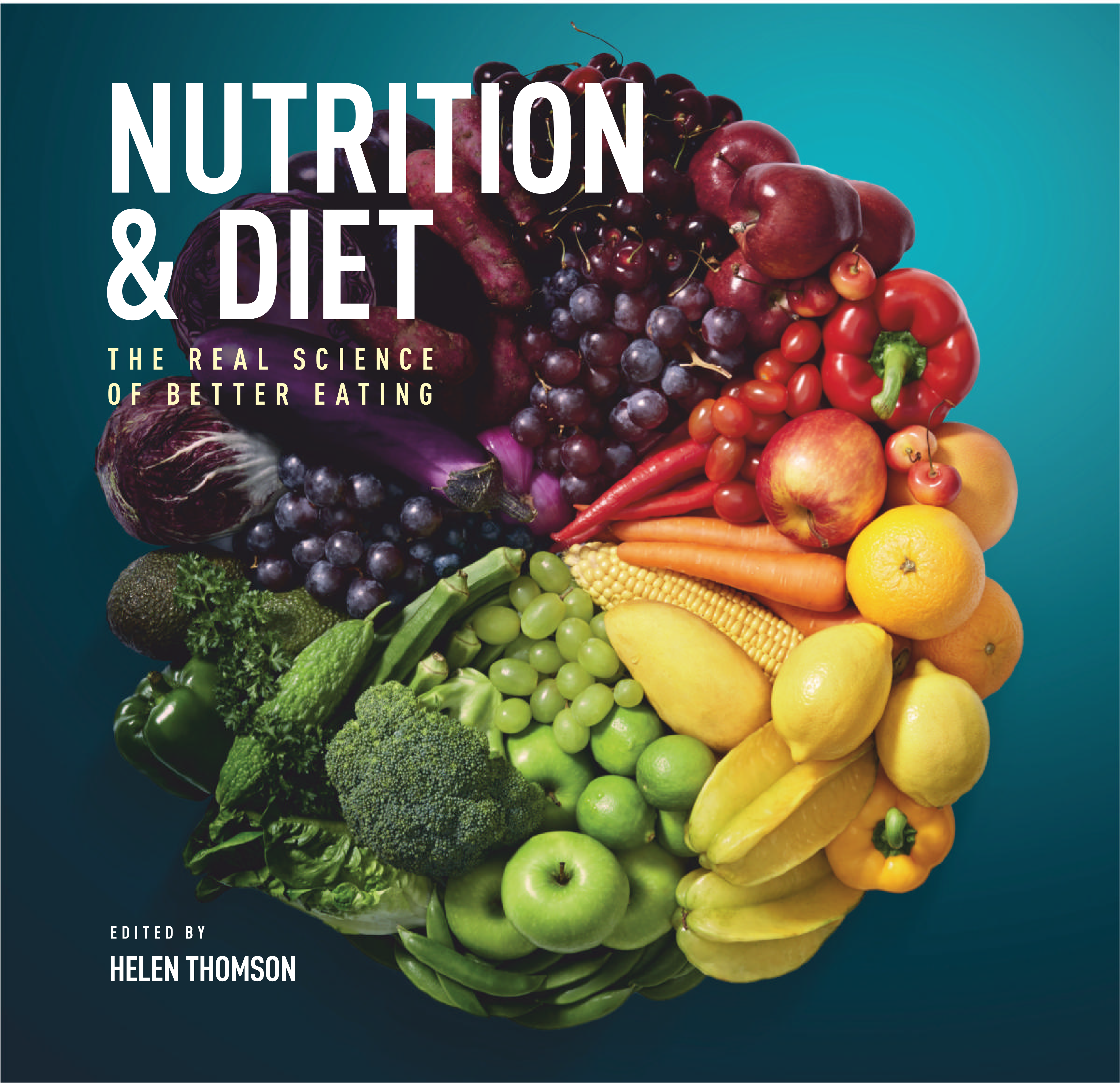
New Scientist

ESSENTIAL GUIDE Nº9

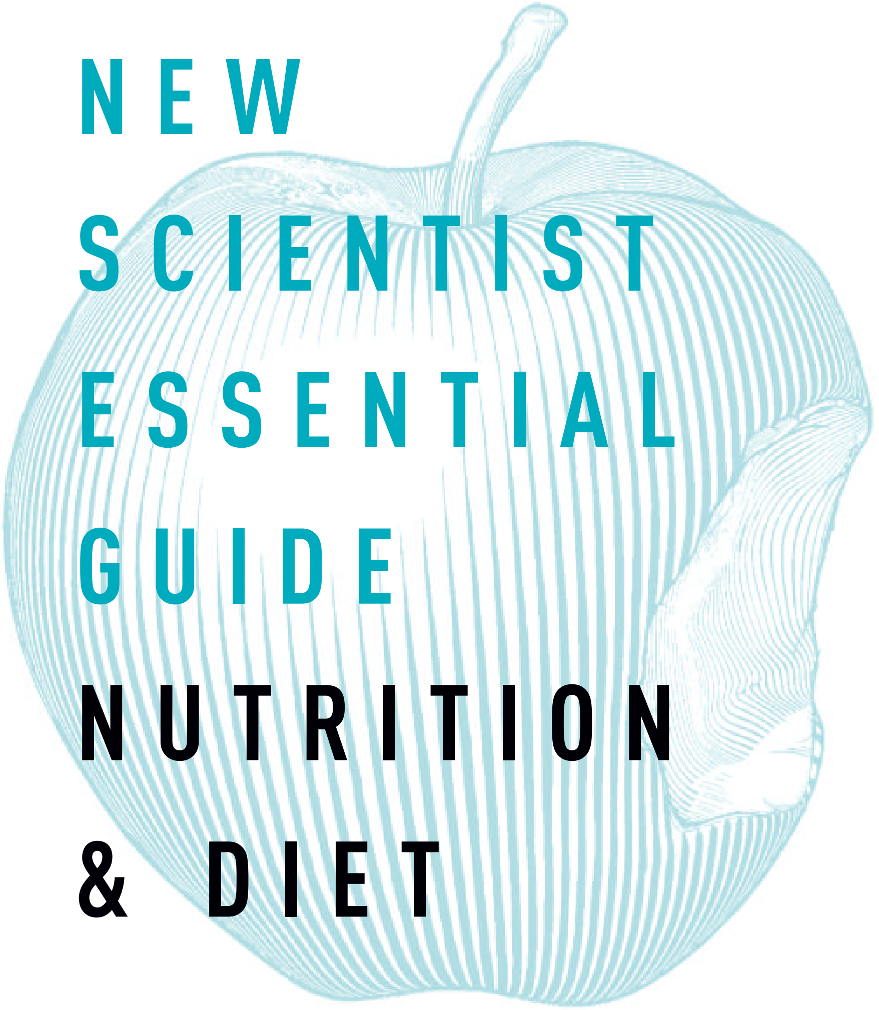
BASICS OF NUTRITION
NUTRITIONAL NASTIES
VITAMINS, MINERALS
AND SUPERFOODS
SUSTAINABLE EATING
THE FUTURE OF FOOD
AND MORE



NUTRITION & DIET

THE REAL SCIENCE OF BETTER EATING

EDITED BY
HELEN THOMSON



NEW SCIENTIST ESSENTIAL GUIDE
NUTRITION & DIET

THERE has never been a greater appreciation than there is now for the importance of what we eat – not just for our physical health, but for our mental health and the health of the planet, too. Even so, with supermarkets, magazines, social media and all manner of celebrities bombarding us with advice on what to eat and how to buy sustainably, you would be forgiven for not having a clue about what should be on your weekly shopping list.

The aim of this latest *New Scientist Essential Guide*, the ninth in the series, is to bypass fashion and faddery and give an overview of what science says about healthy nutrition and a sustainable diet. We start with the origins of the current obesity crisis and working through what is good and bad in our current diets to find how we might eat better now, and how healthy nutrition might look different in the future.

Do tuck in – I hope you find some satisfying morsels within the following pages. Previous editions in the *Essential Guides* series can be bought by visiting shop.newscientist.com; feedback is welcome at essentialguides@newscientist.com. *Helen Thomson*

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CHAPTER 1

BASICS OF NUTRITION

An overabundance of calories in many modern societies has put a firm focus on questions of good diet. But as with anything that has to do with the human body, the issues involved are hugely complex. What we eat is just one of many factors that determine the effect food has on our physical and mental health.

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CHAPTER 2

NUTRITIONAL NASTIES

Modern dietary advice sees the greatest nutritional evil as the overconsumption of saturated fat, leading the epidemic of obesity and heart disease we see today. Yet other elements of our diet have recently come into the frame.

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SUPPLEMENTS AND SUPERFOODS

The presence of certain substances in just trace amounts in our food can make all the difference in warding off disease and helping us live healthier, longer lives. The science behind vitamins and minerals is well-established. Whether that means we need to be regularly popping pills or eating special foods that contain them in particular abundance is another question entirely.

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FOOD AND DIETING

Whether it's cutting carbs, maxing out on protein or simply eating very little at all at times, many diets have been claimed to help us maintain a healthy weight and live longer lives. Figuring out whether any of them work brings us close to chewing up a century or more of orthodoxy – in fact, there may be no such thing as one healthy diet.

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Every food choice we make has an impact on the wider world, whether you eat meat and fish or you're a staunch vegan. Radically changing what we eat is easier said than done, however. How to persuade people to change their diet, and how to produce food more sustainably, remain contentious issues.

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Issues of sustainability and our still-evolving understanding of what constitutes good nutrition mean that what's on our plates in a few decades' time is likely to look very different to today. The question of whether we will find foods produced via new technologies palatable remains.

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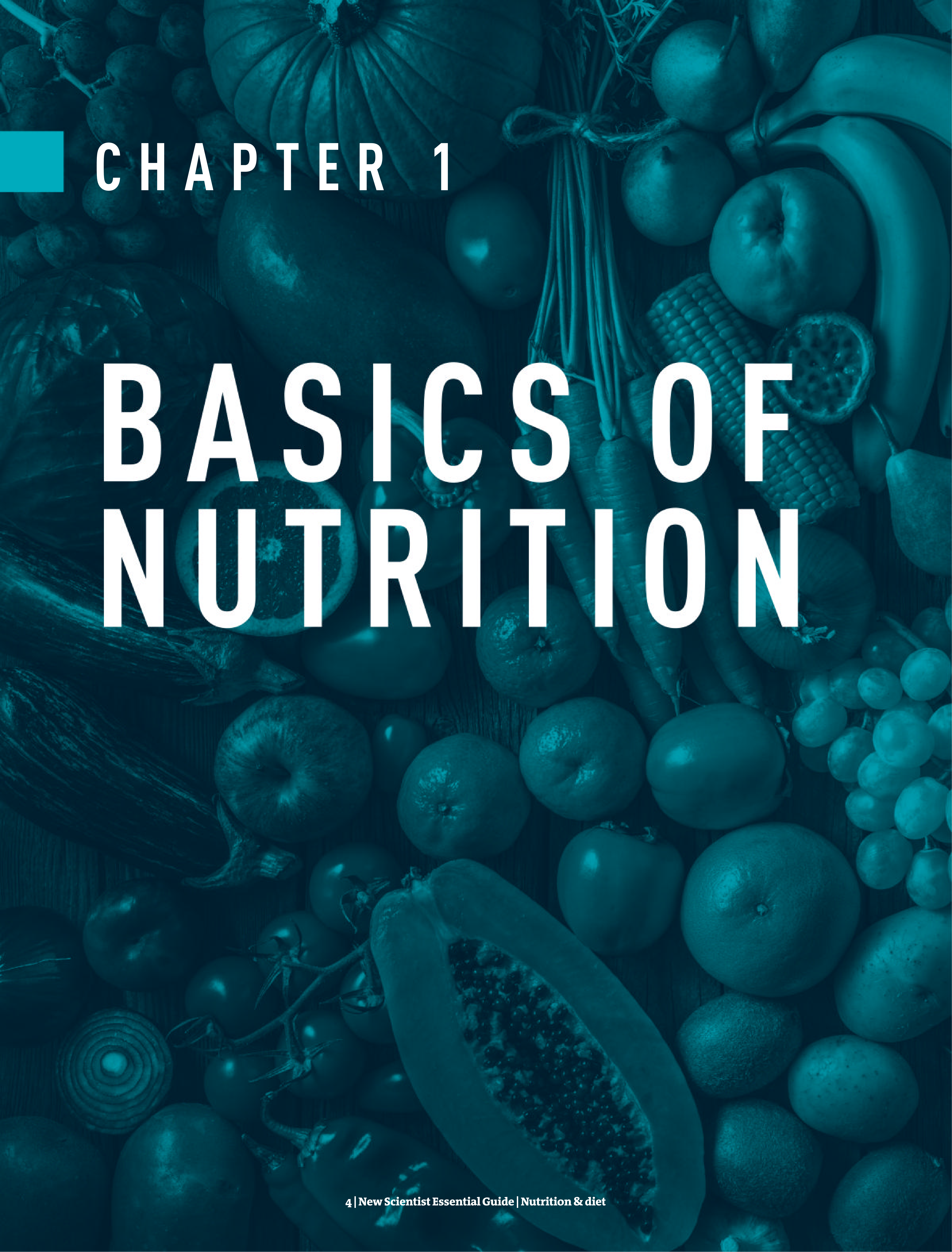
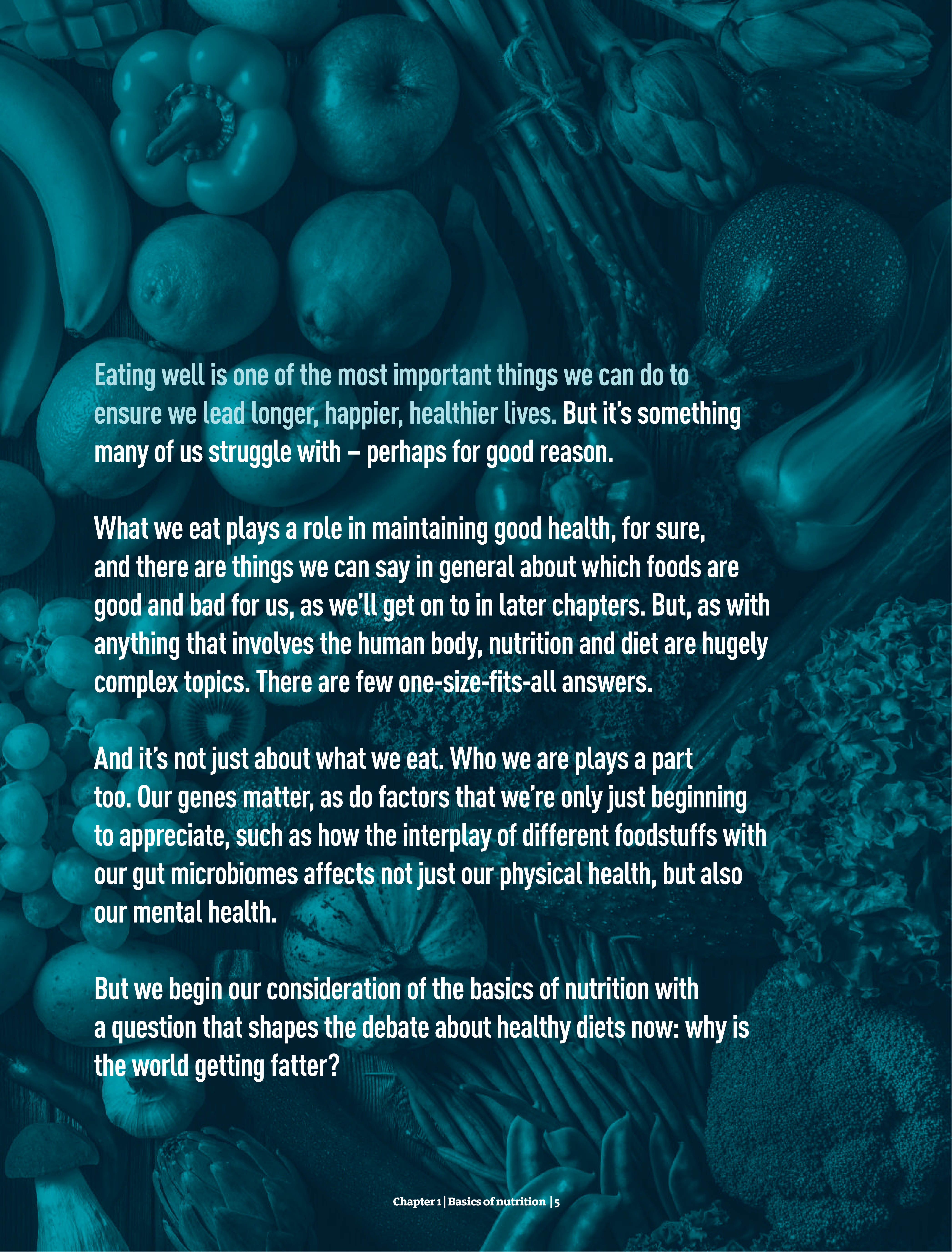


CHAPTER 1

BASICS OF NUTRITION



Eating well is one of the most important things we can do to ensure we lead longer, happier, healthier lives. But it's something many of us struggle with – perhaps for good reason.

What we eat plays a role in maintaining good health, for sure, and there are things we can say in general about which foods are good and bad for us, as we'll get on to in later chapters. But, as with anything that involves the human body, nutrition and diet are hugely complex topics. There are few one-size-fits-all answers.

And it's not just about what we eat. Who we are plays a part too. Our genes matter, as do factors that we're only just beginning to appreciate, such as how the interplay of different foodstuffs with our gut microbiomes affects not just our physical health, but also our mental health.

But we begin our consideration of the basics of nutrition with a question that shapes the debate about healthy diets now: why is the world getting fatter?

THE ORIGINS OF THE OBESITY CRISIS

There's a central point that motivates many concerns about nutrition: most of us are getting bigger, and we know that isn't good for us. But why many nations are seeing a rise in obesity, and what to do about it, are questions that go to the heart of the complexities within nutritional science.

IN NEARLY every country in the world, average weight is on the increase. We are getting fatter for a multitude of reasons. First, and most simply, people are eating more. Abundant, cheap food means average daily calorie intake has gone up in high-income nations, from around 3000 calories a day in the 1960s to 3400 in the past decade, according to the World Health Organization.

Second, we may be eating the wrong sorts of food. For a long time, the chief villain was seen as too much fat in our diet. More recently, attention has shifted more towards sugar and highly processed fast foods, with many doctors citing a shift from eating home-cooked food as an underlying factor in bulging waistlines.

→ Chapter 2 has more on nutritional villains such as fat, sugar and salt

Third and last, people do less manual labour, walk less and spend more time sitting down in front of screens than they used to. Some believe this is the real explanation for rising obesity rates, rather than any dietary factor.

Whatever its cause or causes, we know for certain that obesity is linked to all manner of horrible outcomes, including an increased risk of cancer, heart disease and diabetes. Losing weight and maintaining a healthy body mass index (BMI) lessens our risk of disease, but there is one problem: we don't actually know the best way for people to lose weight and keep it off. ›

PREVIOUS PAGE: MEDITERRANEAN/ISTOCK
RIGHT: SOUTH_AGENCY/ISTOCK

Let's look at exercise first. While exercise seems to help prevent weight gain, studies show it doesn't necessarily help in losing it, perhaps because it is hard to achieve the level of exercise necessary to burn off a significant amount of fat. Exercising more may also make people hungrier. Physical activity seems to have a host of health benefits, including protecting against heart disease and boosting mental health, but weight loss isn't one of them.

↓
There is more on exercise and metabolism on page 14 later in this chapter

Faced with that fact, going on a diet might seem the most obvious option. Then the question becomes: "Which diet?" The answer is far from simple. Consider a recent test of the efficacy of low-fat versus low-carb diets for weight loss. The DIETFITS study put more than 600 people who were overweight on one of these two diets for a year. At the end, the average weight loss was the same in both groups, about 5.5 kilograms, but there was huge individual variation, ranging from much larger losses to significant weight gain.

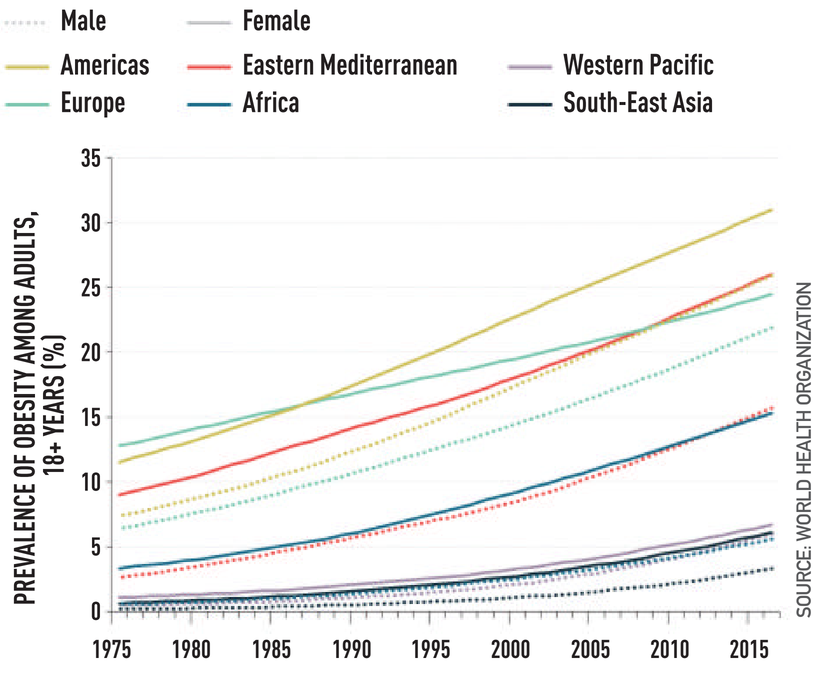
→
Chapter 4 has more on the ins and outs of various weight-loss diets

Advocates of each at least agree that we should be trying to steer people away from highly processed foods, like fast food, ready meals and snacks, which tend to be high in fat, sugar and salt. Given what we know, that seems sensible – although it has to be said that there is very little evidence from randomised controlled trials that eating less processed food leads to weight loss.

There is also the question of how to do the steering. Countless school-based programmes that aim to teach children about exercise and healthy eating have failed to reduce obesity. Public health doctors have long called for tougher measures, such as restricting junk food advertising and the number of fast-food restaurants allowed near schools. Calls to change the way food is taxed so that highly processed food becomes more expensive are controversial, as such foods tend to be cheap and so a shift could disproportionately affect lower-income families.

And while going on a diet might seem like the obvious way to lose weight, and can work initially, keeping it off is much harder. Studies show that most people who lose weight through dieting regain most if not all of it within a year or two, and some end up heavier than when they started. People who attend weight-loss clinics that offer intensive support have somewhat better outcomes, but results are still disheartening.

Obesity has been rising around the world for more than 40 years



SOURCE: WORLD HEALTH ORGANIZATION

Although it may seem extreme, the most effective way to lose weight is to undergo one of the various forms of weight-loss surgery, which shrink the stomach or redirect the gut. These make it harder to overeat, and also mean nutrients aren't absorbed as well.

Randomised trials have shown that such surgeries do help people lose significant weight, and that type 2 diabetes often goes into remission. But it is a major operation that carries risks in addition to the normal concerns about surgery. For example, one study estimates that one in 10 people who have it develop some kind of addiction, such as alcoholism – as if people are transferring a previous "addiction" to food to something else once they can no longer eat the same quantities.

For those who can manage it, however they do so, eating less – and eating more of the right things – comes with benefits beyond those for personal health. There is a growing awareness of the impact some aspects of our modern diets – such as excessive consumption of meat, fish and dairy products like cheese – have on the environment, both in the amount of land they consume and the greenhouse gas emissions they produce directly. All these things mean that a sound grasp of what good nutrition looks like is a question, like the origins of the obesity crisis, of global significance. ∎

→
Chapter 5 has more on sustainable eating

GENETICS AND APPETITE

Our discussion about why we're getting fatter in the previous section missed out a big, complicating factor that we're only just getting to grips with: our genes.

Subtle differences in how we respond to food, driven by our genes, may be why many of us struggle to keep to the weight we want to be.

IT IS no secret that people have big differences in appetite – some eat like birds, others like horses. But only some of these differences reflect our energy needs.

The driving factors for weight gain tend to get oversimplified: studies show that most people still think obesity is down to laziness and gluttony. Others tend to shrug and blame "big bones" or "bad genes".

For many people, that second point might not be too far off the mark. A gene variant that increases a person's obesity risk by 30 to 40 per cent is the strongest genetic predictor of body weight discovered in humans so far. Having just one copy of a particular variant of a gene called CREBRF is associated with a 1.5-unit increase in BMI. For a person who weighs 83 kilograms and is 1.75 metres tall, this is the equivalent of putting on a further 4.6 kilograms.

To investigate how CREBRF promotes weight gain, researchers genetically engineered fat cells in the lab to express the obesity-associated variant. They found that this caused the cells to store more fat and release less energy, as if they are trying to conserve as much fuel as possible. This thriftiness is likely to be advantageous during periods of food scarcity, but to lead to obesity in times of nutritional excess.

In total, genes may be responsible for as much as two-thirds of our variation in weight. But they aren't betraying us in the way many assume. While some like CREBRF do slow down our metabolic rate, leading to a build-up of fat, they are the exception. Most make people fatter in a more insidious way: by subtly affecting how appealing food seems to us, and how quickly we feel full. Not only that, but these genetic

> “Genetic differences in how we respond to food are seen from the first few weeks of life”

effects kick in even from the first few weeks of life.

Measuring how our responses to appealing food and to feelings of fullness can influence weight is something that can be traced back to 2007. That’s when the late British health psychologist Jane Wardle first proposed the idea that genetic differences in appetite raise the risk of overeating in food-rich conditions. This “behavioural susceptibility theory” potentially explained why weight gain can be both highly heritable and highly responsive to environmental influences.

To identify innate differences in appetite and disentangle the impact of genes from all the other factors that can alter our food choices, from social pressures to comfort eating, Wardle and her colleagues turned to twins – specifically, the Twins Early Development Study, a UK survey of more than 15,000 pairs of identical and fraternal twins.

The beauty of such twin studies is that siblings tend to grow up in the same circumstances. That’s important because babies get all sorts of nutritional cues from their mothers – both in the womb and during the first months of life – that can have “a profound effect on developmental systems that regulate appetite”, says Jed Friedman, a molecular nutrition researcher at the University of Colorado, Denver.

At around age 11, children in Wardle’s study were weighed and their parents filled in questionnaires about their eating behaviours, ranking statements such as “My child is always asking for food” and “My child gets full up easily”. This snapshot showed that appetite and weight are linked: children who were more responsive to food and slower to feel full were more likely to carry excess weight. But the researchers couldn’t tell whether the eating traits caused the weight gain or if being overweight was changing children’s habits around food.

The solution was to go right back to the beginning of life, when these things start to emerge. To do so, Clare Llewellyn at University College London has helped run Gemini, the largest ever twin study of childhood weight and appetite, which involves more than 2400 pairs of twins born in England and Wales in 2007. She and her colleagues have found that genetic differences in how we respond to food are apparent from the first weeks of life, and can influence our weight for the rest of our days. “Weight gain is deceptively simple really,” says Llewellyn. “Small differences in appetite on a regular basis can catch up with you.”

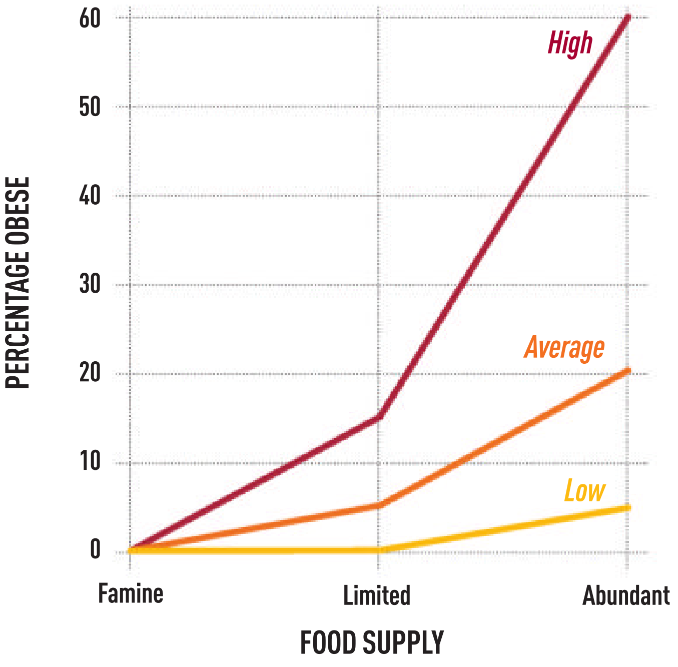
By just a few months of age, this twin study found that babies show clear differences in how they react to milk and how soon they stop feeding once they feel full. By 15 months, twins with strongly divergent eating traits can show as much as a 1-kilogram difference in body weight. That is about 10 per cent of each child’s weight. Project that into adulthood and deep-rooted dining habits may be adding centimetres to your waist.

Gemini revealed it was a heartier appetite during the first three months of life that fuelled early weight gain into toddlerhood, not the other way around. It is the only study to show us that the genetic influences of appetite are visible right from birth.

By the time the Gemini twins turned 2, there was also a distinct divergence in consumption: if you compared two children at opposite extremes of satiety responsiveness, there was a difference of about 37 calories per meal, the equivalent of half a digestive biscuit. Those extra bites quickly add up to more than five extra days’ worth of food consumption per month.

So it isn’t necessarily that children who are overweight are eating fattier or more sugary foods. They may just eat a little bit more at each meal or snack. The same appears to be true for grown-ups.

The amount of available food influences whether people become obese, but models show that a more important factor could be whether you have a high, average or low genetic susceptibility to obesity, which affects what and how much you want to eat



SOURCE: DOI.ORG/10.1016/J.PHYSBEH.2015.07.006

With calorie-rich fare now abundant in kitchens and supermarkets throughout much of the world, people whose genes put them at risk of overeating will consume to excess in a way that wouldn't have been possible in earlier times. Unless we take action to counteract our genetic predilections, waistlines will continue to grow where food is plentiful – and so will the attendant health problems.

This fact has an increasing number of people seeking out a more high-tech solution to their weight problem: DNA tests. Numerous companies sell tests that promise diet and lifestyle recommendations that are purportedly informed by your personal genetic make-up. These usually include a sample of the 200 to 300 gene regions that are tied to weight in some way.

But even if you add up all the known genetic risk factors, they collectively account for maybe 5 per cent of the variation in body weight. How can that be, if, as twin studies have shown, up to two-thirds of this difference may be down to heritable factors? It is either that we haven't identified all the genes at play yet or that they affect behaviour in a more complicated way than we currently understand.

Some experts are worried about the pendulum swinging away from the misconception that overweight people lack willpower to a deterministic fatalism that there is nothing to be done as the problem lies in your genes. The truth is harder to swallow: it isn't a level playing field. Some people just have to work harder not to gain weight.

That brings us to the big question. If our genes predispose us to eat more than our body needs, what can we do about it?

There might be a way to nudge individuals who are overweight in the right direction, without explicitly lecturing them about eating habits, says Claudia ›

WHAT CAUSES MALNUTRITION?

While obesity is a growing public health crisis in many parts of the world, hunger and malnutrition have been far from banished. Nine children die every minute because their diet lacks essential nutrients, according to humanitarian NGO Médecins Sans Frontières. In fact, malnutrition is the single greatest threat to the world's public health, with 178 million malnourished children across the world. If we could eliminate malnutrition, we would prevent 35 per cent of child deaths globally, according to the UN children's agency UNICEF.

Food insecurity – not having enough of the right stuff to eat – obviously plays a huge part. But it seems there is more to the issue than that. One new avenue of research suggests that a person's risk of developing severe malnutrition may come down to the microbes in their guts.

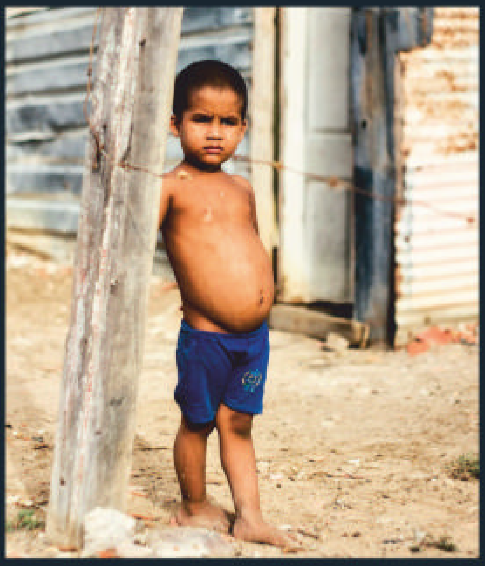
Working in southern Malawi, Jeffrey Gordon at Washington University in St Louis, Missouri, and his colleagues identified sets of identical and non-identical twins in which one child had a severe form of malnutrition called kwashiorkor, thought to be caused by a lack of protein, and the other did not, despite their shared genetics and diet. Gordon's team took faecal samples from three sets of twins and transplanted them into the guts of mice, which were then fed a typical nutrient-poor Malawian diet. All of the mice lost some weight, but some lost significantly more – and more quickly – than others. Further investigation showed that these mice had all received a faecal sample from children with kwashiorkor.

The finding strongly hinted that the mice had picked up a kwashiorkor-like condition from the microbes within the faecal implant, so the researchers studied the rodents' gut flora. They found higher than normal levels of bacteria associated with illnesses such as inflammatory bowel disease.

The results suggest pathogenic microbes may heighten the problems of malnutrition in some children. There is a lot of work revolving around obesogenesis – how, given a standard diet, one set of bugs might make more calories available than another set. But the other side of this coin is that particular bugs might restrict calorie availability and exacerbate a poor diet.

A second study examined how children with kwashiorkor respond when given nutrient-rich diets. The team found that the children were significantly less likely to become malnourished once the dietary treatment had ended if they were given a course of antibiotics along with the diet.

Together, the studies point towards a future in which programmes to prevent food insecurity go hand in hand with microbial concoctions tailored to guard against infections and treat specific conditions.



This child in Venezuela displays symptoms of kwashiorkor, a severe form of malnutrition

NELSON BASTIDAS/ALAMY STOCK PHOTO

Hunot, a dietician at the University of Guadalajara in Mexico. She gave adults a questionnaire about their eating habits, then offered email tips tailored to participants' food-related foibles – be it emotional eating, consuming too quickly or feasting past the point of fullness. "We could give them accessible and simple information that spoke to their own personal traits," says Hunot. If the results suggested someone was very responsive to food, for instance, she might suggest keeping certain foods out of the house or avoiding the table of treats at work.

Another strategy is to build up resistance over time: if chocolate is your weakness, buy a small bar and carry it around, but don't eat it. The idea is that, over time, you will become less tempted to indulge. Feedback from the 32 people who have tried the intervention so far has been positive, says Rebecca Beeken, an obesity researcher at University College London who collaborated with Hunot. "People felt empowered by the knowledge that they could be genetically predisposed to find it harder," she says.

But to truly tackle a proclivity for overeating, the twin studies seem to suggest that interventions have to start much earlier – perhaps even during the first months of life. A common refrain in parenting books and even on government websites is that you can't overfeed a breastfed baby, but Llewellyn's data suggests this isn't completely true.

Ian Paul, a paediatrician at Penn State Children's Hospital in Hershey, Pennsylvania, advocates "responsive parenting". This basically involves paying attention to your child's needs and learning the difference between cries of hunger, overstimulation and tiredness, for instance, and then tailoring your responses accordingly instead of turning to food as a default solution. Paul and his colleagues studied about 300 first-time mothers, half of whom were trained in responsive parenting, the other half in infant safety. Those in the first group were half as likely to have a 1-year-old who was overweight and one-tenth as likely to have a 2-year-old who was obese.

Part of responsive parenting is limit-setting, says Paul. This simply means being sensible about portion sizes if you have a child who will overeat given the chance. That might sound simple, but it is a lot harder to do in practice when your baby is crying. "It's a very tricky topic," says Llewellyn. "No parent wants to be told what to do."

This is why some believe that less of the burden of addressing the obesity crisis should be placed on the individual. Theresa Marteau, director of the Behaviour and Health Research Unit at the University of Cambridge, thinks the UK government should start placing restrictions on the portion sizes of unhealthy foods. Economic analyses suggest that this would make a massive dent in the country's public health burden from obesity. Smaller portions, packages and tableware could reduce daily energy intake by significant amounts, says Marteau.

Ultimately, though, science may have failed to tackle the obesity epidemic because the way we respond to food varies so much from person to person. We will need more than randomised controlled trials of different diets and other interventions to solve this problem. The answer may lie in personalised nutrition – integrating individuals' metabolisms and microbiomes into our studies of obesity, diet and healthy living. That, however, is a science very much in its infancy.

→
For more on personalised nutrition, turn to page 68

Perhaps there are reasons to celebrate our different responses to food, too. Danielle Reed at the Monell Chemical Senses Center in Philadelphia, Pennsylvania, likens the propensity for overeating to genetic differences in vision. Just as someone who sees colours more vibrantly might spend longer at the Louvre than the average person, someone who has the genes to savour the "sensory loveliness of food" might take those extra bites even after they are full. If you struggle to refuse that extra slice, perhaps it is some consolation that you might be able to taste in technicolour and better appreciate all the glorious flavours. ▮

EXERCISE AND METABOLISM

There are many myths about the effect of exercise on our bodies: that it naturally increases our metabolism, boosting calorie expenditure and expediting weight loss, for example.

Recognising these myths may help explain why our exercise routines sometimes don't have the effects we want – but that doesn't mean exercise isn't a good thing.

IT IS the bedrock belief of pretty much every workout routine: exercise more and burn more calories. In the short term, it is correct. You burn energy while you are exercising, and if you start a new workout routine, you will burn more calories – at least in the beginning.

But recent research shows just how dynamic and adaptive our metabolisms can be. Take studies of the Hadza people in northern Tanzania, who lead a physically demanding lifestyle subsisting on wild plants and game, with no guns, machines or domesticated animals. Hadza men log 19,000 steps each day while hunting and gathering wild honey, and women log 12,000 steps while collecting wild tubers and berries, often with children on their backs. This is about five times more physical activity each day than the average for Western lifestyles, and yet Hadza men and women burn the same number of daily calories as sedentary office workers in industrialised populations.

It isn't just the Hadza: farmers and foragers in other small-scale societies, with equally high daily workloads, have the same daily calorie expenditures as people in high-income countries. It seems our bodies work to keep the daily number of calories burned within a narrow range, regardless of lifestyle.

New workout regimes tend to be subjected to the same metabolic adjustment. Daily expenditures measured for participants in exercise studies routinely increase at the beginning, but those gains diminish

PS-I/ALAMY STOCK PHOTO

The Hadza people in Tanzania burn few calories, despite being hugely physically active

over time. Our bodies adapt so that within a few months, the daily energy we burn is only marginally higher – and sometimes exactly the same – as before we started working out more. The boost is a bust, for most of us.

Even those who do manage to increase the amount of energy they burn through exercise typically still find it hard to lose weight. A recent review of 61 exercise studies, totalling more than 900 participants, lays out the grim evidence that will be familiar to many.

Weight loss often starts off well at the beginning of a new exercise regime, but it fades over time so that, a year or so later, the weight lost is a vanishing fraction of what we would expect from all the calories burned through working out.

In one of the longer trials, men and women in the US burned 2000 calories per week during supervised exercise sessions for 16 months. After nine months, the men had lost around 5 kilograms, after which their weight plateaued. Women in the study lost no weight over the entire 16 months. Neither the men nor the women lost what we would have expected based on their exercise workload, despite the fact that their daily energy expenditures had edged up slightly.

The reason for this is frustratingly simple: when you burn more calories, you eat more calories. The complex systems working subconsciously to regulate your hunger and satiety do an exceptional job of matching energy intake to expenditure. As a result, the amount of weight you can expect to lose from exercise alone over the course of a year is a paltry 2 kilograms or less.

But while exercise might not change the number on your bathroom scales, that isn't necessarily what it is for. Humans evolved as hunter-gatherers, and a heavy dose of physical activity was an inescapable part of the daily routine for more than 2 million years. There are good reasons why peoples such as the Hadza avoid heart disease and diabetes, despite the fact that they burn the same amount of calories as sedentary people. Regular exercise keeps our hearts healthy, our muscles strong and our minds sharp, especially as we age.

Intriguingly, recent studies suggest that the very metabolic adjustments that frustrate weight loss might be a big reason why exercise is so good for us. Our bodies respond to increased daily activity by reducing the energy expended on other tasks. For example, immune systems quieten down, reducing inflammation, which is important because it is a risk factor for cardiovascular disease and a range of other health problems.

People who exercise regularly also respond to stressful events with smaller surges of the stress hormones cortisol and adrenaline, reducing their risk of stress-related disease. Finally, comparisons of oestrogen and progesterone in women and testosterone in men commonly show reduced levels among adults in physically active populations. These reductions don't affect fertility but have been linked to a lower risk of prostate, ovarian and breast cancer.

Ultimately, exercise won't make us thin, but it will give us longer lives. ▮

THE ROLE OF THE MICROBIOME

There is a deep-rooted connection between our guts and our brain, which potentially gives us a way to improve not just our physical health, but also change our mood through our food. Recently, scientists have begun to home in the central role of an entity that's part of us and yet not part of us: our gut microbiome.

WILDPIXEL/ISTOCK

FOR many people, sweet treats are the way to happiness. Doughnuts are particularly difficult to resist. Recent studies show why: treats that are high in both carbs and fats trigger a supercharged amount of activity in our brain's reward centre that releases the "feel-good" chemical dopamine.

The researchers behind this study think that we may have separate systems in the brain to evaluate fatty or carb-heavy foods. If both get activated at the same time, this tricks the brain to produce a larger amount of dopamine – and a bigger feeling of reward – than there should be based on the food's energy content.

This could be because when the human brain evolved, our hunter-gatherer ancestors ate a diet consisting mainly of plants and meat, and never encountered food that is high in both carbs and fat. The brain is used to seeing one signal at a time, but modern food is tricking the system. The finding fits with studies on rodents that revealed they can regulate their calorie intake when given food containing only fat or carbohydrate, but overeat and gain weight when given access to foods containing both.

But there are other ways that food can influence your mood. Most recently, researchers have turned to the link between your brain and your gut microbiome.

From the moment we are born, we acquire and nurture an internal ecosystem of symbiotic bacteria and other microbes. These organisms outnumber our own cells, though not by 10 to one as is often claimed. An average human body is made up of about 30 trillion human cells and 38 trillion microbial ones. By mass, we absolutely dwarf our companions: a 70-kilogram human contains just 200 grams of microbe.

But they punch well above their weight, and none more so than the ones that live in our gut. Our gut microbiota do huge amounts of work digesting food that the biochemical products of the human genome can't break down on their own. They are, in fact, the principal determinant of how we respond to food.

Our gut microbiome can influence our brain through several routes. Gut bacteria send signals up the vagus nerve directly to the brain; they influence immune cells in the gut, which produce a range of chemicals that affect the brain; and they create chemicals that travel in the blood, some of which get through the blood-brain barrier and into the brain directly.

What we eat also influences which gut bacteria are present in our bodies. Research shows that people who lack certain gut bacteria, such as *Dialister* and *Coprococcus*, are more likely to experience depression. This doesn't necessarily mean that lacking these bacteria causes depression. It could be that people who have depression eat differently, for instance, and that this changes their gut flora. However, other studies support the idea that you can influence your risk of depression with what you eat.

For instance, there are studies in people that suggest that gut bacteria can affect hormones and brain chemicals like GABA, which has a calming effect on some areas of the brain. Another study showed that giving people a mix of bacteria for six weeks reduced their symptoms of depression compared with a placebo group.

Yet it isn't clear which kinds of bacteria we need to promote good mental health, or in what quantities. Many people take prebiotics, which are dietary fibre that feed gut bacteria, and probiotics, which are the actual bacteria. The jury is still out here, however. Megan Rossi at King College London says prebiotics are found in thousands of foods that we would normally eat, so most people don't need them. However, there is good evidence that people with certain conditions will benefit from taking a probiotic supplement. One example is when you are taking antibiotics – in this case, probiotics can significantly reduce your risk of antibiotic-associated diarrhoea, which affects about 30 per cent of people.

Rather than take a supplement, Rossi suggests trying to include a wide range of plant-based foods in your diet, such as whole grains, nuts, seeds, legumes and fruit. Studies suggest that people who have at least 30 plant-based elements in their weekly diet have a more diverse range of bacteria in their gut, which is associated with better weight management, better heart health and better mental health.

One easy change is to get a packet of mixed seeds and put a teaspoon on your breakfast – that's four extra elements right there. Or get a packet of mixed leaf salad, rather than a single lettuce, because each type of lettuce has different plant chemicals that feed different bacteria. ∎

WHAT IS TASTE, ANYWAY?

You might find it slightly distasteful, but your body has taste buds all over the place – even in your testicles, if you have them. Understanding the strange sensation of taste shows it is about more than pleasure: it is hardwired into our basic biology and we are only just learning how to harness it.

WHEN we eat something, what we experience is its flavour, a combination of taste, smell and other sensory inputs. But while the scent of food is derived from a palette of 10,000 volatile chemicals, taste is much simpler. Millennia of experience and centuries of data identified four familiar, traditional, fundamental tastes: salt, sweet, bitter and sour. These primary tastes can be combined to beautiful effect, but not broken down further.

An early hint of other primary tastes came in 1908 when a Japanese scientist extracted a compound called monosodium glutamate (MSG) from seaweed. Kikunae Ikeda at Tokyo's Imperial University considered MSG the embodiment of what he called umami, which can be translated as "delicious taste". People often describe it as the savoury quality of meats and cheese.

The following year, a company called Ajinomoto started selling MSG as a flavour enhancer. Today, most of us consume MSG every day in our diets. Foodies across the world may now be familiar with umami, › but whether it really is a primary taste has been debated for decades. Settling arguments like this involves painstaking evidence. One critical exercise is identifying receptors in our taste buds that recognise the specific taste molecules involved. Other important tests include conducting tastings with groups of people who don't have the genetic instructions to make the receptor in question. If they can't discern a taste, it is probably a member of the primary club.

Attempts to find receptors for the assumed primary tastes were boosted in 2000 with the first publication of the human genome. By 2011, scientists had used it to hunt down receptors for salt, sweet, sour, bitter and umami. This, combined with an abundance of tasting data, has convinced most people that there are five primary tastes.

But it doesn't end there. In recent decades, a sixth candidate entered the ring – kokumi, an entirely different concept to umami. The term kokumi, which roughly translates as "dense, full-bodied flavour", was originally used to refer to a property of cooked garlic. In 1990, Ajinomoto researchers identified several compounds containing sulphur that they thought were responsible. But their taste tests showed up something strange. Although Ajinomoto has found a receptor for kokumi in taste buds, volunteers who tried the compounds reported that they had no taste of their own. Instead, they gave food what the firm calls "continuity, mouthfulness and thickness".

Ajinomoto hopes that kokumi could be a way of returning the thick, luxurious texture that foods can lose when fat is stripped out. The firm showed in a taste test that adding a kokumi compound to a low-fat peanut butter made it seem thicker to people eating it. Kokumi dishes also require less salt and sugar than their counterparts without it. A 2009 study showed that compounds known to strongly stimulate the receptor also increase the kokumi intensities that people experience, while molecules that block the receptor suppress kokumi's flavour enhancement.

But can you call something that has no taste a basic taste – and if so, what does it consist of? The answer may lie in a more subtle contributor to flavour: touch. We already know that touch is involved in flavour perception. For example, pain and temperature-sensing receptors connected to the trigeminal nerve, responsible for sensation in the face, can be irritated to give peppermint's coolness and chilli or mustard's heat. Kokumi may be acting on the kind of touch receptors that respond as food flows around in the mouth.

A touch effect would explain why kokumi still works even when people's noses are pegged. It would also widen the potential uses for kokumi ingredients. Perhaps if you enhanced flavour with kokumi, it would help older people who have lost their sense of smell and become malnourished through loss of interest in eating. It might also improve aeroplane food, which seems lousy in part because our sense of smell is less effective at the lower air pressure found in cabins.

We may be back to five primary tastes – but more are possibly in the works. A metallic taste, like you get if you suck the blood from a cut, is the sixth candidate, and good old fat is the seventh. Tasting studies haven't adequately identified whether fat is a primary taste or a modifier of others yet. But there is a plausible candidate for a fat taste receptor and it is important in our gut as well as our mouth.

The interest in looking elsewhere for taste receptors stems from the search for the sensor that triggers a rise in blood insulin when we eat glucose. In 2007, Robert Margolskee at the Monell Chemical Senses Center in Philadelphia, Pennsylvania, and Josephine Egan at the US National Institutes of Health found taste receptors in cells in the first section of our small intestine that are involved in stimulating insulin production.

This discovery showed that taste receptors aren't just for consciously tasting food, but can also send messages through our bodies without involving our brains. And they aren't just in guts. Salt taste receptors have been found in cells from a cow's neck. Researchers have also found taste receptors in the lungs and – weirdly – the testicles. Some speculate that here they might be detecting nutritional status to avoid the body wasting valuable energy making sperm when food is scarce. But no one knows for sure.

Companies are already trying to exploit these findings, developing dietary supplements that target the sweet, umami and bitter receptors in the gut. The aim is to get them to dispatch more fullness signals, helping people feel full for longer – and so lose weight ▮

WHY MOST NUTRITIONAL ADVICE IS WRONG

Before we get down into the nitty-gritty of what kinds of food are good and bad for us in the following chapters, we should preface things with a big caveat. Nutritional science is complex, as we have said – and most of what we think we know about it is probably the product of flawed studies.

IT IS often easy to wonder whether we should believe anything we read about food – when we see the latest headline claiming that eating eggs increases our risk of a heart attack, say, or that a piece of dark chocolate a day keeps the doctor away. Advice about what to eat (or not to eat) seems to change every week.

Many of the apparent contradictions stem from the fact that most food studies are of a certain kind that makes them easier to carry out, but more likely to lead to false conclusions. The gold standard for human medical trials is a randomised controlled trial, or RCT. Here, doctors ask a random half of their subjects to take a medicine, while the rest take identical dummy pills so no one knows who is taking what. If those that take the real drug end up in better health, there is a good chance the medicine was responsible.

This kind of study is hard to do for food. Few would agree to change their diet for years on a doctor's random say-so, and it would be hard to keep secret what they are eating. Instead, nutrition scientists usually observe what people eat by asking them to fill out food diaries, then track the health of participants.

The big problem with these "observational" studies is that eating certain foods tends to go hand in hand with other behaviours that affect health. People who › eat what is considered an unhealthy diet – more fast food, for instance – tend to have lower incomes and unhealthy lifestyles in other ways, such as smoking and taking less exercise. Conversely, eating supposedly healthy foods correlates with higher income, with all the benefits that brings.

For example, even if blueberries don't affect heart attack rates, those who eat more of them will have fewer heart attacks, simply because eating blueberries is a badge of middle-class prosperity.

Researchers use statistical techniques to try to remove such "confounders". But no one knows for certain which confounders to include, and picking different ones can change results dramatically. For instance, vitamin E supplements have been shown in different studies to reduce death rates, have no effect or even raise deaths, depending on what confounders are used.

→ Chapter 3 has more on vitamins, minerals and dietary supplements

Only a tiny fraction of published nutrition studies are large, good-quality RCTs. The rest are mainly observational studies, small or poorly designed trials, opinion pieces or reviews that summarise the results of other papers, with all their potential flaws. Another source of error is publication bias: studies that show interesting results are more likely to get published than those that don't.

Even national dietary guidelines are based on this potentially flawed and insufficient evidence base. For instance, there are no randomised controlled trials that show fruit and vegetables or fibre affect mortality or rates of heart attacks and cancer. Despite all the urging that we should aim for five portions of fruit and veg a day – or maybe seven or even nine, depending on who you listen to – no trial has shown that doing so makes us live longer.

It would be unfair to conclude that nutrition science has taught us nothing, though. It was thanks to dietary studies that we identified the role of vitamins in good nutrition – for instance, that rickets was caused by a lack of vitamin D, as well as the benefits of folic acid (vitamin B_9) supplementation for pregnant people.

And, to point out the obvious, we don't need an RCT to know that eating too much cake can make us seriously overweight, which physically strains the circulatory system and joints, or that not eating enough fibre makes us constipated. What's more, while research on controversial diets like fat versus carbs may not be perfect, it is still churning up interesting new discoveries that might one day see us reach a satisfying conclusion. Nutritional science is also helping us delve into the reasons why a lot of us simply can't stop overeating, and assisting us in overcoming these unhealthy habits.

→ For more on various diets, see chapter 4

There are ways to separate the wheat from the chaff when it comes to nutritional advice, too. First, be careful about taking one study on its own and assuming that it is the be-all and end-all – look to the broader evidence. Second, try to focus on randomised trials rather than small, observational studies. And lastly, appreciate that a lot of nutritional changes make very small differences to your health, so don't expect one diet tweak to be the answer to everything. ▮

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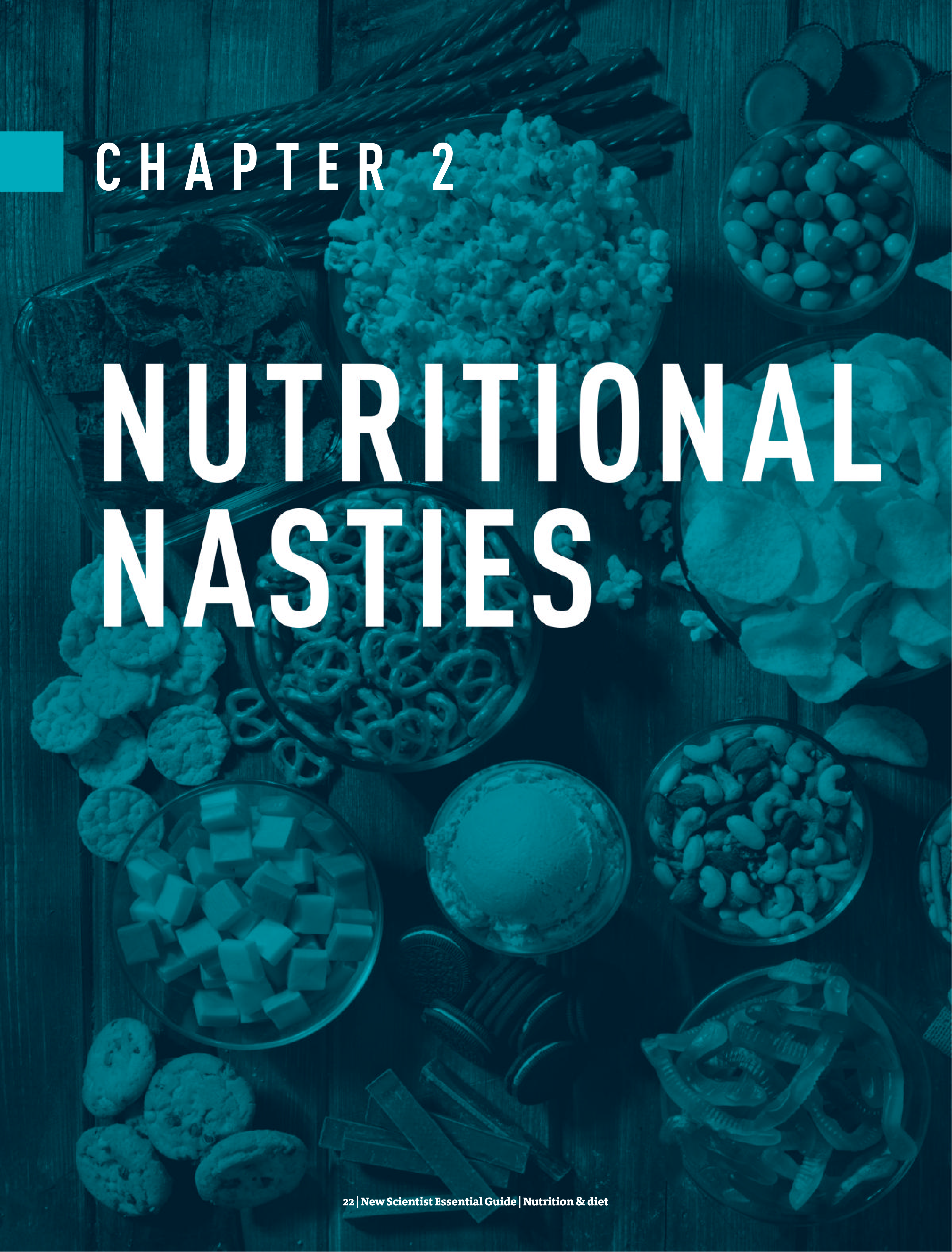


CHAPTER 2

NUTRITIONAL NASTIES

The complexities in nutritional science begin even when you look at the big picture. For most of the past 50 years, the gospel has been that the best diet to follow is what is sometimes known as a "Mediterranean" diet.

This is characterised by plenty of fresh fruit and vegetables, grains, nuts, fish and unsaturated fat in the form of olive oil – and above all low quantities of the ultimate bogeyman, saturated fat. That probably isn't far wrong as a guide to healthy eating, but as levels of obesity have continued to rise in more recent years, questions have been raised as to whether saturated fat is as bad as it has been made out to be.

Is sugar the villain instead? Or salt? Or a combination of all three in the vast quantities of processed "junk" food that many modern diets contain? Read on to find out the latest thinking.

THE TRUTH ABOUT SATURATED FAT

The anchoring fact of most official nutritional advice is that we should aim to reduce our consumption of saturated fats. Their undesirable effects supposedly include raising levels of "bad" cholesterol in our blood and increasing our risk of heart disease. But recent studies suggest the truth is far more nuanced – on all counts.

PREVIOUS PAGE: LAURI PATTERSON/ISTOCK

THERE is a famous scene in Woody Allen's film *Sleeper* in which two scientists in the year 2173 are discussing the dietary advice of the late 20th century. "You mean there was no deep fat, no steak or cream pies or hot fudge?" asks one, incredulous. "Those were thought to be unhealthy," replies the other. "Precisely the opposite of what we now know to be true."

We aren't quite in this territory yet, but the idea that saturated fat is bad for your heart is no longer the uncontested truth it once was.

The right answer matters. According to the World Health Organization, cardiovascular disease is the world's leading cause of death, killing more than 17 million people annually, about a third of all deaths. It predicts that by 2030, 23 million people will succumb each year. An estimated 81 million people in the US are living with cardiovascular disease. The healthcare bill is a small fortune.

For a long time, the official guidance in the US for adults was that no more than 30 per cent of total calories should come from fat, and no more than 10 per cent from saturated fat (see diagram below). UK advice is roughly the same. That is by no means an unattainable target: an average man could eat a whole 12-inch pepperoni pizza and still have room for an ice cream before busting the limit. Nonetheless, adults in the UK and US manage to eat more saturated fat than recommended.

That is still less than it was. From the 1950s to the late 1970s, fat accounted for more than 40 per cent of dietary calories in the UK. It was a similar story in the ›

The total recommended daily
energy intake for men is about

2500 calories

No more than

750 calories should come from fat

No more than

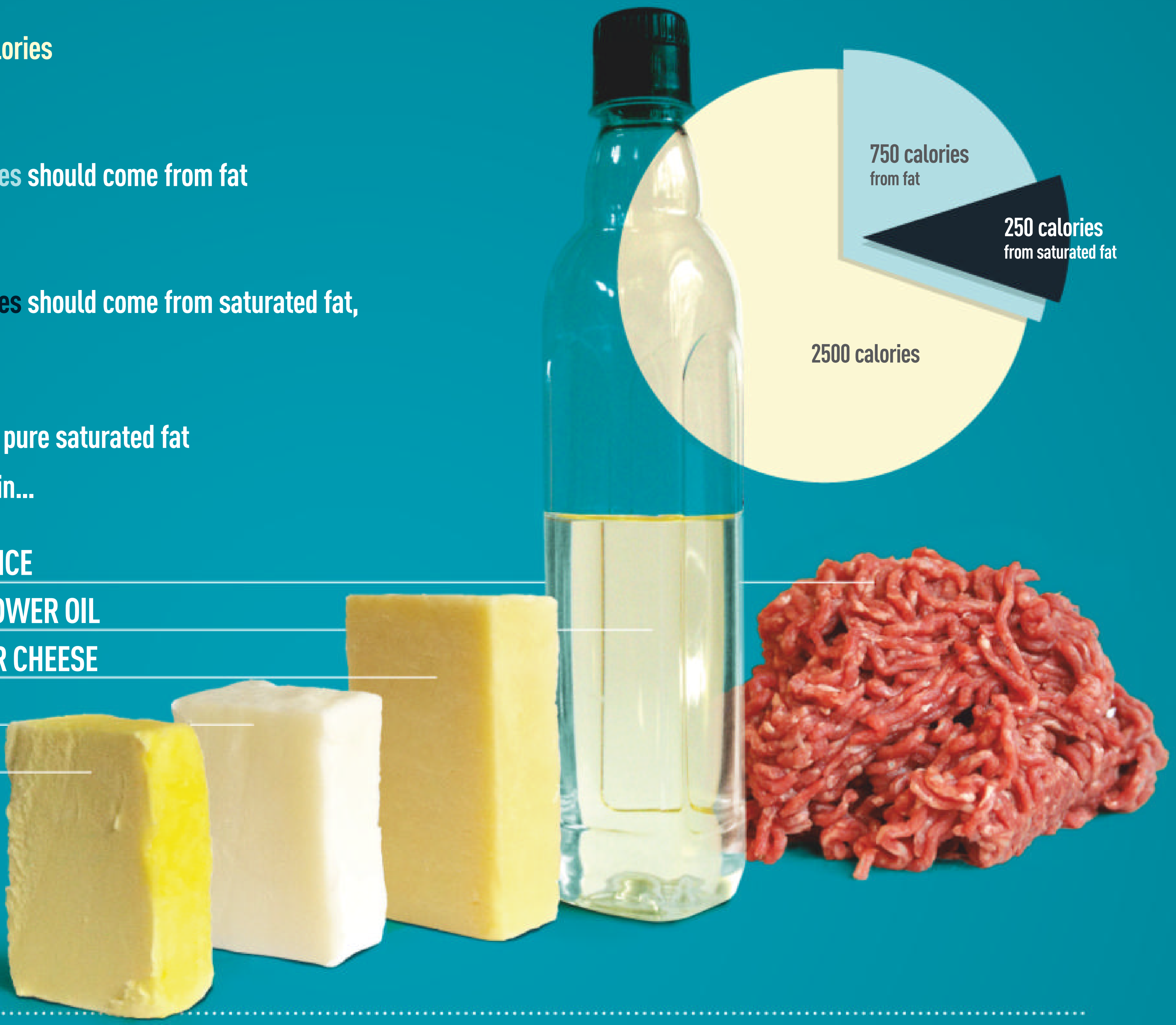
250 calories should come from saturated fat,

equal to

28 grams of pure saturated fat

which is found in...

600G BEEF MINCE
280ML SUNFLOWER OIL
133G CHEDDAR CHEESE
72G LARD
55G BUTTER



Lesser of two evils?

Some things you probably think of as being low in saturated fat are not - and vice versa

An avocado contains
as much saturated fat as
five packets of crisps

(~3g)

A large latte* contains
more saturated fat than a
cream doughnut

(6.6g vs 6.2g)

*16 fl.oz, (473 ml), whole milk

Nuts are richer
in saturated fat
than steak

(9% vs 3.5%)

“Fat consumption has gone down, but obesity and associated diseases haven’t”

WHAT IS A FAT?

Fats are complex biomolecules that play various roles in the body, including energy storage and as components of cell membranes. A fat molecule is made up of three fatty acids bound to a molecule of glycerol. This unit is known as a triglyceride. There are dozens of different types of fatty acid, all with different properties, including whether they are saturated or unsaturated.

WHAT DOES SATURATED/UNSATURATED MEAN?

The bulk of a fatty acid is a long string of carbon atoms with hydrogen atoms attached. In a saturated fatty acid, this chain does not have any carbon-carbon double bonds, meaning it has the maximum possible number of hydrogens: it is “saturated”. Unsaturated fatty acids have at least one double bond.

Triglycerides containing only saturated fatty acids are also called saturated; those with one or more double-bonded acids are unsaturated. As a rule, the more unsaturated a fat, the better it is for you – though this orthodoxy is being challenged.

WHAT ABOUT ANIMAL AND VEGETABLE FATS?

Fats from animals tend to be saturated, while those from vegetables are usually unsaturated. But this is only a rough guide. Meat, eggs and dairy contain unsaturated fats, while vegetables also contain saturated fats. Some vegetable fats – notably palm oil, coconut oil and the cocoa butter used in chocolate – are higher in saturated fat than beef dripping or lard. In short, both animals and plants contain saturated and unsaturated fat.

IS CHOLESTEROL A FAT?

Strictly speaking, no. But it is a vital link between dietary fat and heart disease. Unlike saturated fat, cholesterol is almost exclusively found in animal products: meat, fish, seafood, milk and eggs. Cutting the cholesterol in your diet doesn’t have much direct effect on blood cholesterol levels, but can help indirectly because cutting down on cholesterol-rich foods will usually reduce your saturated fat intake.

ARE SOME FATS MORE FATTENING THAN OTHERS?

No. A gram of unsaturated fat contains just as much energy as a gram of saturated fat (about 9 calories, compared with about 4 calories in a gram of sugar). So in terms of calories, there are no “good” and “bad” fats.

US. As warnings began to circulate, people trimmed back on foods such as butter and beef. The food industry responded, filling the shelves with low-fat cookies, cakes and spreads.

Deaths from heart disease have also gone down in Western nations. In the UK in 1961, more than half of all deaths were from coronary heart disease; in 2009, less than a third were. But medical treatment and prevention have improved so dramatically that it is impossible to tell what role, if any, changes in diet played. And even though fat consumption has gone down, obesity and its associated diseases haven't.

When you eat fat, it travels to the small intestine where it is broken down into its constituent parts – fatty acids and glycerol – and absorbed into cells lining the gut. There, they are packaged up with cholesterol and proteins and posted into the bloodstream. These small, spherical packages are called lipoproteins, and they are what allow water-insoluble fats and cholesterol (together known as lipids) to get to where they are needed.

The more fat you eat, the higher the levels of lipoprotein in your blood. And that, according to conventional wisdom, is where the health problems begin.

Lipoproteins come in two main types: high density and low density. Low-density lipoproteins (LDLs) are often simply known as "bad cholesterol", despite the fact that they contain more than just cholesterol. LDLs are bad because they can stick to the insides of artery walls, resulting in deposits called plaques that narrow and harden the vessels, raising the risk that a blood clot could cause a blockage. Of all types of fat in the diet, saturated fats have been shown to raise bad cholesterol levels the most. (Consuming cholesterol has surprisingly little influence: the reason it has a bad name is that it is found in animal foods that also tend to be high in saturated fat.)

High-density lipoproteins (HDLs), or "good cholesterol", on the other hand, help guard against arterial plaques. Conventional wisdom has it that HDL is raised by eating foods rich in unsaturated fats or soluble fibre such as whole grains, fruits and vegetables. This, in a nutshell, is the lipid hypothesis, possibly the most influential idea in the history of human nutrition.

The hypothesis traces its origins back to the 1940s, when a rising tide of heart attacks among middle-aged men was spreading alarm in the US. At the time, this was explained as a consequence of ageing. But Ancel Keys, a physiologist at the University of Minnesota, had other ideas.

He noted that heart attacks were rare in a lot of countries bordering the Mediterranean nations. The typical diets of these countries – at least in popular depiction, which has also been influenced by Keys's findings – are characterised by a lot of fresh fruit, vegetables, grains, nuts and fish. They also go easy on the meat and dairy, but have lashings of olive oil – and very little saturated fat. Convinced that there was a causal link, Keys launched the pioneering Seven Countries Study in 1958. In all, he recruited 12,763 men aged 40 to 59 in the US, Finland, the Netherlands, Italy, Yugoslavia, Greece and Japan. The participants' diets and heart health were checked five and 10 years after enrolling.

Keys concluded that there was a correlation between saturated fat in food, raised levels of blood lipids and the risk of heart attacks and strokes. The finding was supported by other research, notably the Framingham ›

Heart Study, which tracked diet and heart health in a town in Massachusetts. In light of this research and the rising death toll – by the 1980s, nearly a million people in the US were dying from heart attacks each year – health authorities decided to officially push for a reduction in fat, and saturated fat in particular. Official guidelines first appeared in 1980 in the US and in 1991 in the UK, and have stood largely unaltered ever since.

Yet the voices of doubt have been growing for some time. In 2010, scientists pooled the results of 21 studies that had followed 348,000 people for many years. This meta-analysis found "no significant evidence" in support of the idea that saturated fat raises the risk of heart disease. Another meta-analysis in 2014 revisited the results of 72 studies involving 640,000 people in 18 countries. "Current evidence does not clearly support guidelines that encourage high consumption of polyunsaturated fatty acids and low consumption of total saturated fats," it concluded. "Nutritional guidelines… may require reappraisal."

In essence, the research found that people at the extreme ends of the spectrum – that is, those who ate the most or least saturated fat – had the same chance of developing heart disease. High consumption of unsaturated fat seemed to offer no protection.

These studies generated a lot of controversy among nutrition researchers. Outside of academia, the conclusion was greeted with gusto. Many commentators interpreted it as a green light to resume eating saturated fat. But is it? Did Keys really get it wrong? Or is there some other explanation for the conflict between his work and the many studies that supported it, and the two recent meta-analyses?

Even as Keys's research was starting to influence health advice, critics were pointing out flaws in it. One common complaint was that he cherry-picked data to support his hypothesis, ignoring countries such as France that had high-fat diets but low rates of heart disease. The strongest evidence in favour of a low-fat diet came from Crete, but it transpired that Keys had recorded some food intake data there during Lent, a time when Greek people traditionally avoid meat and cheese, so he may have underestimated their normal fat intake.

The Framingham research, too, has its detractors. Critics say that it followed an unrepresentative group of predominantly white men and women who were at high risk of heart disease for non-dietary reasons, such as smoking.

More recently, it has also become clear that the impact of saturated fat is more complex than was understood back then. Ronald Krauss at the University of California, San Francisco, has long researched the links between lipoproteins and heart disease. He was involved in the 2010 meta-analysis and is convinced there is room for at least a partial rethink of the lipid hypothesis.

Krauss points to studies suggesting that not all LDL is the same, and that casting it all as bad was wrong. It is now widely accepted that LDL comes in two types – big, fluffy particles and smaller, compact ones. It is the latter kind, says Krauss, that is strongly linked to heart-disease risk, while the fluffy type appears to be a lot less worrying. Crucially, eating saturated fat boosts fluffy LDL, says Krauss. What's more, there is some research suggesting that small LDL gets a boost from a low-fat, high-carbohydrate diet, especially one rich in sugars.

For Jeremy Pearson, a vascular biologist and associate medical director at the British Heart Foundation, which part-funded the 2014 meta-analysis, it doesn't mean we should throw out the orthodoxy surrounding saturated fat. He points to hard evidence from studies in animals, where dietary control is possible to a degree that it isn't in people. They repeatedly show that high levels of saturated fat lead to high LDL and hardened arteries, he says.

“The effect of reducing saturated fat depends on what replaces it”

So how do we explain the contradiction? “I guess what that means is that in free-living humans, there are other things that are usually more important regarding whether you have a heart attack or not than the balance of saturated and unsaturated fat in your diet,” says Pearson. Factors such as a lack of exercise, alcohol intake and body weight may simply overshadow the impact of fat.

Certainly, the debate can’t be divorced from the issue of overall calorie intake, which rose in the US and many other countries in the three decades from the 1970s. The result was rising numbers of people who were overweight, and being overweight or obese raises the risk of heart disease. Another key factor might be what people now eat instead of saturated fat. “The effect of reducing saturated fat depends on what replaces it,” says Walter Willett at the Harvard School of Public Health. “We consciously or unconsciously replace a large reduction in calories with something else.”

The problem, as some see it, is that the something else is usually refined carbohydrates, especially sugars, added to foods to take the place of fat. A review in 2009 showed that if carbohydrates were raised while saturated fat was cut, the outcome was a raised heart-disease risk. This plays to the emerging idea that sugar is the real villain.

↓
The next section has more on sugar

Then there are trans fats. Created by food chemists to replace animal fats such as lard, they are made by chemically modifying vegetable oils to make them solid. Because they are unsaturated, and therefore “healthy”, the food industry piled them into products such as cakes and spreads. But it later turned out that trans fats cause heart disease. All told, it is possible that the meta-analyses simply show that the benefits of switching away from saturated fat were cancelled out by replacing them with sugar and trans fats.

Meanwhile, science continues to unravel some of the intricacies of fat metabolism, which could also help account for the confusing results. One promising avenue is that not all types of saturated fat are the same. The 2014 meta-analysis, for example, found clear indications that different saturated fatty acids in blood are associated with different coronary risks. Some saturated fats appear to lower the risk; some unsaturated ones increase it.

A study from 2012, meanwhile, found that while eating lots of saturated fat from meat increased the risk of a heart attack or stroke, equivalent amounts from dairy actually reduced it. Krauss also cites studies showing that eating cheese doesn’t raise bad cholesterol as much as eating butter, even when both have identical levels of saturated fat.

The problem is that studies of single nutrients have a fundamental flaw, says Marion Nestle, a professor of nutrition at New York University. “People do not eat saturated fat,” she says. “They eat foods containing fats and oils that are mixtures of saturated, unsaturated and polyunsaturated fats, and many other nutrients that affect health and also vary in calories. So teasing saturated fat out of all that is not simple.”

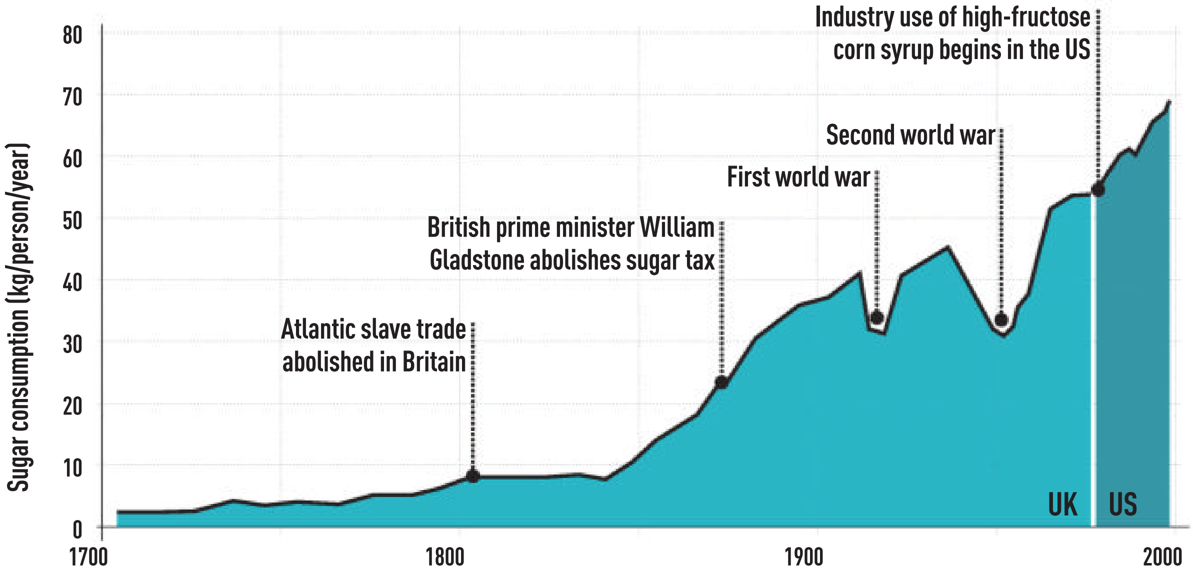
The only way to rigorously test the various hypotheses would be to put some people on one kind of diet and others on another for 20 years or more. “Doable? Fundable? I don’t think so,” says Nestle.

So while dietary libertarians may be gleefully slapping a steak on the griddle and lining up a cream pie with hot fudge for dessert, the dietary advice of the 1970s still largely stands – for now. In other words, steak and butter can be part of a healthy diet. Just don’t overdo them. ■

SUGAR ON TRIAL

Sugar is increasingly touted as public health enemy number one. Some people see it as perhaps the major driving force behind obesity, heart disease and type II diabetes – and a toxic, addictive and above all unnecessary evil to boot. But is sugar really so sickly sweet?

Sugar consumption per person in the UK and US has been steadily rising



SOURCE: AMERICAN JOURNAL OF CLINICAL NUTRITION, VOL 86, P901

IMAGINE you are sitting at a table with a bag of sugar, a teaspoon and a glass of water. You open the bag and add a spoonful of sugar to the water. Then another, and another, and another, until you have added 20 teaspoons. Would you drink the water?

Even the most sweet-toothed child would find it unpalatably sickly. And yet that is the amount of sugar you are likely to eat today, and every day – usually without realising it.

"Sugar" refers to a large class of sweet-tasting, energy-dense carbohydrates. The simple sugars glucose, fructose and galactose, and the more complex sucrose and lactose, are the most familiar to us.

Whatever form it takes, sugar has become a large and growing part of our diets in recent decades. If you eat processed food of any kind, it probably contains added sugar. Three-quarters of the packaged food sold in US supermarkets has had sugar added to it during manufacturing. You can find it in sliced bread, breakfast cereals, salad dressings, soups, cooking sauces and many other staples. Low-fat products often contain a lot of added sugar.

When nutrition scientists talk about the adverse effects of sugar, they aren't fretting about sugars found naturally in foods such as fruit and vegetables, or the lactose in milk. Instead, they are worried about added sugar, usually in the form of sucrose (table sugar, made of a molecule of glucose and a molecule of fructose bonded together) or high-fructose corn syrup.

Our early ancestors would have been totally unfamiliar with these refined forms of sugar, and until relatively recently, sugar was a rare and precious commodity. Only in the 1700s, after Europeans had introduced sugar cane to the New World and tied its cultivation to slavery, did it become a regular feature of the Western diet. In 1700, the average English household consumed less than 2 kilograms of table sugar a year. By the end of the century, that amount had quadrupled (see diagram above).

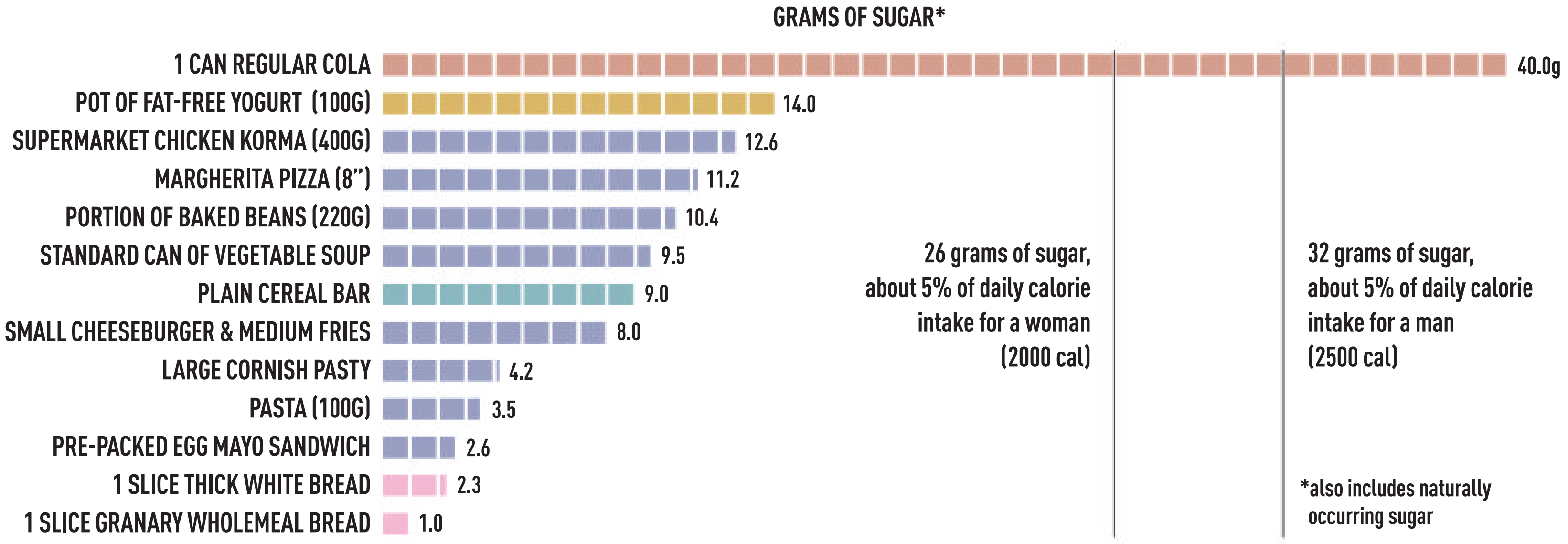
The upward trend has continued largely unbroken ever since. Between the early 1970s and the early 2000s, adults in the US increased their average daily calorie intake by 13 per cent, largely by eating more carbohydrates, including sugar. In 1996, the average US adult swallowed 83 more calories per day from added sugar than in 1977. Today, yearly sugar consumption in the US is close to 40 kilograms per person – more than 20 teaspoons a day.

The sugar rush has many causes, but one of the most important was the invention of high-fructose corn syrup in 1957. It is a gloopy solution of glucose and fructose that is as sweet as table sugar but has typically been about 30 per cent cheaper.

Once this source of sweetness was available, food manufacturers added it liberally to their products (see charts overleaf). "Because hunger is no longer an important factor in most developed countries, what ›

KLENOVA/ISTOCK

The World Health Organization is likely to recommend that no more than 5% of calories should come from added sugar. Large quantities are hidden in processed foods...



*also includes naturally occurring sugar

can make people eat more?" says Serge Ahmed, a neuroscientist at the University of Bordeaux, France. "Food pleasure. And what creates food pleasure? Sugar."

Not all scientists see eye to eye on the health effects of sugar, but there is one point on which most agree: we don't actually need it. Luc Tappy, a physiologist at the University of Lausanne in Switzerland, sums it up. "You cannot live without essential fats. You cannot live without protein. It's going to be difficult to have enough energy if you don't have some carbohydrate. But without sugar, there is no problem," he says. "It's an entirely dispensable food."

All that unnecessary sugar adds calories to our diet, so it is no surprise that the rise in consumption coincided with the rise of obesity and related problems, such as type 2 diabetes. In 1960, around one in eight US adults was obese; today, more than a third are. Since 1980, obesity levels have quadrupled in the developing world to nearly 1 billion people. One study found that for every additional 150 calories' worth of sugar available per day in a country, there is an associated 1.1 per cent rise in diabetes.

Robert Lustig, an endocrinologist at the University of California, San Francisco, is one of the most outspoken critics of sugar's role in our diets. Described by some of his peers as an anti-sugar evangelist, Lustig's main beef is with fructose, a simple sugar found naturally in fruit, but is also a component of sucrose and high-fructose corn syrup.

The case against fructose is built on the fact that, unlike glucose, it doesn't play an essential role in human metabolism. (That isn't to say we need to eat glucose; complex forms of carbohydrate, such as starch, supply all the glucose our metabolisms need when they are broken down inside our bodies). Our ancestors would have encountered fructose in fruit, but in nothing like the quantities we eat today, so part of the argument is that our bodies simply aren't adapted to deal with it.

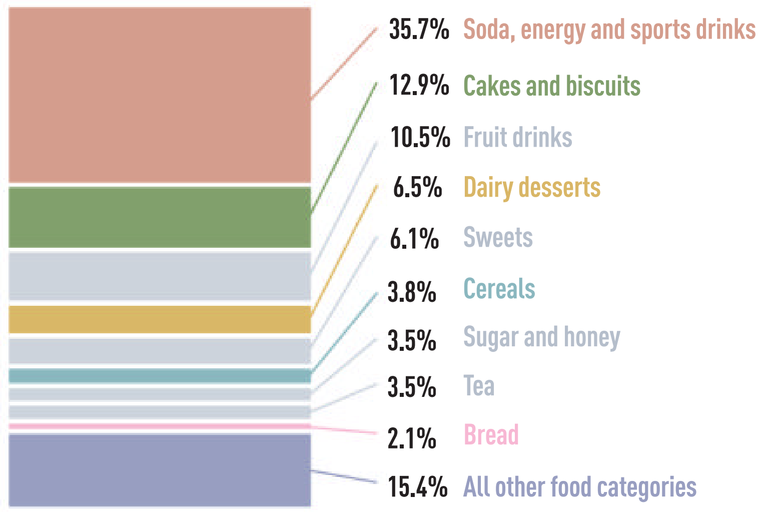
To begin with, fructose is almost exclusively metabolised by the liver. When we eat a lot of it, Lustig and others say, much of it is converted into fat. Fat build-up in the liver can lead to inflammation and scarring, and can progress to cirrhosis. Fatty liver has also been linked to insulin resistance, a precursor to diabetes.

Fructose is converted into energy, but Lustig claims that, unlike glucose breakdown, this produces lots of oxygen radicals – dangerously reactive chemicals that attack our bodies and cause ageing.

Mopping these up requires antioxidants, but how many you get often depends on the quality of your diet. "People who can't afford better food don't get the antioxidants. That's one of the reasons why people in the lower socioeconomic strata get sicker on the same dose of sugar," says Lustig.

What's more, unlike glucose, fructose isn't regulated by insulin. This hormone keeps blood glucose levels stable and spurs the production of leptin, the hormone that lets you know when you are full. Fructose doesn't affect leptin production; one small study even suggests it ups the level of its counterpart, ghrelin, the hormone

…meaning the average US adult eats more than 100 grams a day, with most coming from soft drinks (100g sucrose: ~400 calories)



SOURCE: WHO, US DEPARTMENT OF HEALTH & HUMAN SERVICES

that makes you feel hungry. In other words, fructose encourages overeating.

Finally, eating lots of fructose has been shown in both animal and human studies to boost levels of triglycerides in the blood, which increase the risk of hardened arteries and heart disease.

Many nutrition scientists remain unconvinced. A number of studies have failed to find evidence that fructose is uniquely harmful – though these have been criticised because their authors received funding from food and beverage companies. More credibly, in 2012, Tappy reviewed the case against fructose. He concluded that while there is cause for concern in people who already have a metabolic disease or are at risk of developing one, there is no evidence that fructose is the sole cause, or even the main cause, of these diseases. But the case remains open. “There are many unanswered questions,” says Tappy.

Another sinister claim against sugar is that it warps eating habits by altering brain chemistry to make us want more. For several years, neuroscientists have found it useful to compare energy-dense foods with addictive substances such as cocaine – at least in a metaphorical sense – because it equips them with the language to discuss their habit-forming properties.

Several studies in rats have shown that a burst of sweetness affects the reward system in the brain in a similar way to cocaine. One study even gave cocaine-addicted rats the option between cocaine and sugar water. “Most turned away from the drug for the sweet reward,” says Ahmed, who ran the study.

In humans, foods that are high in fat and sugar – called “hyperpalatable” foods – are known to trigger our reward systems by boosting dopamine levels much as addictive drugs do. And there is research suggesting that most people with conditions such as binge-eating disorder display similar psychological characteristics to people with substance abuse problems.

Alarmed by reports of sugar’s dangers, around a decade ago, the World Health Organization’s Nutrition Guidance Expert Advisory Group began carrying out a review of the evidence on sugar with a view to making some recommendations. As part of that process, Lisa Te Morenga, a researcher in human nutrition at the University of Otago in New Zealand, reviewed the research on the relationship between sugar and body weight in 2013. She concluded that it wasn’t necessarily eating too much sugar that was making us fat, but eating too much of everything. If total calorie count was controlled for, people didn’t get any fatter when more of those calories came from sugar.

When Te Morenga looked at studies that more closely replicate food choices in real life, however – that is, when participants weren’t held to precise calorie counts – those who ate a lot of sugar tended to consume more calories overall and gained more weight. And the most important source of sugar was one that has been high on the list of obesity campaigners’ concerns for years: sugary drinks. This was yet more evidence that sweetened drinks really do cause weight gain – which is the strongest reason to point the finger at sugar.

Why does it matter if we consume calories in liquid rather than solid form? Think of it this way. It takes about 2.5 oranges to make a glass of juice. But drinking a glass doesn't make you feel as full as eating two-and-a-half oranges. That is because the fibre in the fruit makes you feel fuller for longer.

As Te Morenga puts it, "all sugar-sweetened drinks really do is contribute calories to the diet" – but without making you full. This is partly because fructose, which can make up 65 per cent of the sugar in drinks from soda fountains, doesn't activate the fullness hormone leptin.

This lack of satiety from sugary drinks makes it possible to consume many more calories at a sitting than you would otherwise. Having a sugary drink with a meal, for example, doesn't make you eat less. This lack of satiety in exchange for calories seems to have long-term consequences. Several epidemiological studies have linked the consumption of sugary drinks with increased risk of obesity, type 2 diabetes and heart disease.

Te Morenga isn't convinced that sugar is the uber-villain of our health woes, but is confident that the way sugary foods are marketed makes us eat more than we need. "Maybe people should take more personal responsibility," she says. "But we're letting food companies spend millions of dollars to convince people to buy their products – or that soft drinks are a perfectly normal thing to have with a meal."

As the dangers of overconsumption of sugar become more apparent, more and more places in the world are weighing up or implementing taxes on sugary drinks and the like. Meanwhile, for individuals, the advice is quite simple: try to reduce how much sugar you are consuming. Above all, avoid sugary drinks. "It's the easiest thing to do," says Tappy.

Of course, critics of efforts to curb sugar intake will counter that if you simply eat well and exercise, sugary drinks and snacks can be reasonable indulgences. That's true, so far as it goes. But there is another simple truth about sugar: however much you might want it, you really don't need it. ▮

SALT: KILLER OR SCAPEGOAT?

For many of us, salt is a routine part of our diet. And yet this mineral that we so casually sprinkle onto our food has a reputation as a killer in excessive quantities – if not immediately, then over time. It probably deserves such notoriety.

SALT – or, more accurately, its constituent ions sodium and chloride – is a vital nutrient. Sodium and chloride help maintain fluid balance, and sodium is one of the ions that nerve cells use to create electrical impulses.

The typical food available to our hunter-gatherer ancestors was low in salt, so we have evolved an exquisite system for detecting it in our diet. One of our (at least) five types of taste bud is dedicated to salt, the only one tuned to a single chemical. Unlike energy, our bodies can't readily store salt, so we are experts at hanging on to it, largely through a recycling unit in the kidneys. It is possible to survive perfectly well on very little salt.

Until recently, most humans ate no salt other than what was naturally in their food, amounting to less than half a gram a day. Pure salt only entered the food chain around 5000 years ago when people in China discovered it could be used to preserve food. Salt has since played a leading role in human history, and become one of the world's most valued commodities.

Although we no longer have to rely on salt to keep food from spoiling, our appetite for it is undiminished. Most people eat much more salt than they need. While US dietary guidelines set an adequate intake of 3.75 grams a day, the average Westerner eats about 8 grams; in some parts of Asia, 12 grams is the norm.

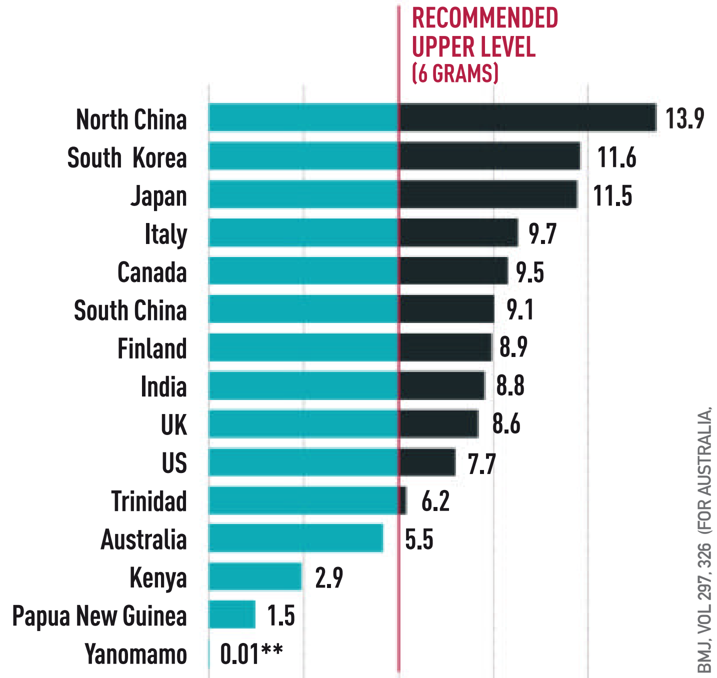
Despite a widespread belief that we have an innate liking for salt, this appetite appears to be learned. Research shows that people living in some traditional societies with no access to pure salt, such as highlander communities in Papua New Guinea, find it repulsive. If they move to the city, however, they quickly take to it. As with chilli and caffeine, it seems we can learn to love salt's intrinsically aversive flavour.

And like an addictive drug, the more you eat, the more you crave, as salt receptors on the tongue become desensitised by overuse. Once in this habituated state, unsalted foods taste bland and uninteresting. It can take several weeks of salt withdrawal for taste preferences to return to normal.

It doesn't help that today's diet is full of salt (see diagram overleaf). Around three quarters of the salt we eat is added to food before it even reaches our plates, not only in the obvious culprits like cured meat and smoked fish, but also concealed in breakfast cereal, biscuits, cheese, yoghurt, cake, soup and sauces. Even bread is surprisingly salty.

There is a multitude of reasons why processed food is so laden with salt. As well as prolonging shelf life, it makes cheap ingredients taste better and masks the bitter flavours that often result from industrial cooking processes. It can be injected into meat to make it hold more water, thus allowing water to be sold for the price of meat. It improves the appearance, texture and even the smell of the final products. And it makes you thirsty, boosting sales of drinks.

National average salt intake (grams of salt per day)

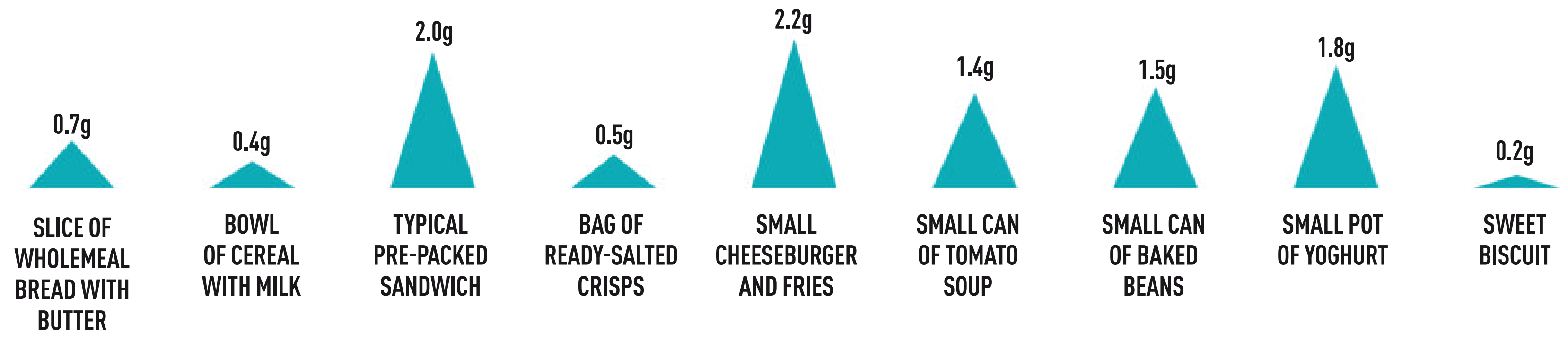


** an Indigenous people of the Amazon on the Brazil-Venezuela border who have the lowest recorded salt intake in the world

SOURCE: BMJ, VOL 297, 326 (FOR AUSTRALIA, WORLD ACTION ON SALT AND HEALTH)

MALERAPASO/ISTOCK

Typical salt content of some common processed foods. Recommended upper level per day is 5.0 - 6.0 grams



This effortless consumption of salt horrifies doctors. Our kidneys can excrete some excess salt, but even so, people who consistently eat more than about half a gram a day – that is, practically all of us – build up excess sodium. To keep fluid concentrations stable, our bodies retain extra water. "We're all sloshing around with a litre or a litre and a half compared with what we would be if we were on our evolutionary salt intake," says Graham MacGregor, a professor of cardiovascular medicine at the Wolfson Institute of Preventive Medicine in London.

An inevitable consequence of this excess fluid is a rise in blood pressure. Exactly how isn't clear. Nor is the reason why some people are more sensitive to it than others. But the fact that it occurs is uncontroversial, and it is the effect on blood pressure that causes problems. High blood pressure is one of the main risk factors for cardiovascular disease; even small increases raise your risk of having a stroke. "Everything that lowers blood pressure works. There's no argument," says MacGregor.

For this reason, salt reduction has become one of the most important public health targets in the West. Dietary guidelines vary, but generally recommend eating no more than 5 to 6 grams of salt a day. And these levels are far from ideal – they are merely what is considered realistic in a world awash with salt.

There is no lack of evidence that eating less salt is good for us. One approach is to look for a link between how much salt people eat when left to their own devices and their rates of heart attacks and strokes. Over the years, many such studies have been done. In 2009, cardiologist Francesco Cappuccio at the University of Warwick, UK, pooled all the data and found a strong relationship between a salty diet and cardiovascular disease.

Another way is to intervene directly in people's diets – take two groups of people, get one of them to eat less salt for a while and see what the outcome is. These trials take more work than so-called observational studies, but several have been done. The biggest managed to get thousands of people to cut down on salt by about 2 grams a day for up to four years, and saw a 25 per cent fall in cardiovascular disease.

Alternatively, you can look at whole countries, taking the before-and-after approach. Fifty years ago, northern Japan had one of the world's biggest appetites for salt – an average of 18 grams a day per person – and shockingly high numbers of strokes. The government implemented a salt-reduction programme and, by the late 1960s, average salt consumption had fallen by 4 grams a day and stroke deaths were down by 80 per cent. Finland, another salt-guzzling nation, achieved similar results in the 1970s.

There is some counter-evidence. One meta-analysis found that while cutting salt lowers blood pressure, levels of certain hormones and lipids in the blood increase, which could theoretically raise cardiovascular risk. But many of the studies included in the analysis lasted just a few days and involved big salt reductions. MacGregor accepts that sudden and steep salt reduction can lead to counterproductive hormonal changes, but says that modest reductions, say of 6 to 8 grams, don't. "There's no evidence whatsoever that a modest reduction does any harm."

One lesson from these latest studies is that headlines can be misleading; the devil is in the detail. That is why the salt reducers talk about the "totality of the evidence". Nutrition science is notoriously hard. You need large numbers of people to detect the outcome of small dietary changes and there are so many confounding factors that sometimes paradoxical results pop up.

"Nutrition is not black and white," says Susan Jebb at the UK Medical Research Council's Human Nutrition Research unit in Cambridge. "It's not about one definitive trial, it's about the totality of the evidence. In this case, the balance of evidence strongly supports reductions in salt."

"We have much stronger evidence for salt than we do for fat, for the benefits of eating fruit and vegetables or losing weight," says MacGregor. "There has never been a randomised controlled trial of cigarette reduction. Should we not have done anything about cigarettes?" ▮

IS JUNK FOOD REALLY THAT BAD FOR US?

Fat, sugar, salt. One form of food has them all in abundance: ultra-processed junk food. We are eating more of it than ever, but if there is one thing nutritionists tend to agree on, it's that we should avoid it.

That said, what it is in junk food that makes it so bad for us isn't yet clear. This has made it difficult to know what exactly we should be avoiding and why.

FACTORY-MADE food comprises between 50 and 60 per cent of the average person's calorie intake in the UK. Large studies in the past two decades looking at the links between the consumption of ultra-processed food and health certainly paint a bad picture, with people who eat the most being at a higher risk of obesity and conditions such as heart disease and diabetes. One study even found that the more ultra-processed food that has undergone extensive factory processing (see "Defining junk", page 39) in a person's diet, the higher their risk of early death.

Importantly, while nutrition research is notorious for its contradictory results, this doesn't seem to be the case for ultra-processed foods.

So far, so bad. But, as ever, there are confounding factors. We know, for instance, that eating more junk food also correlates with having a low income, which brings many health disadvantages.

←
Turn back to page 19 for more on problems with nutrition studies

To solve this problem, Kevin Hall, a physiologist at the US National Institutes of Health, and his colleagues enrolled 20 participants into a randomised trial. He asked volunteers to stay on site for the four-week duration of the experiment so that every morsel passing their lips could be analysed. For two weeks, › they ate snacks and meals that were mainly ultra-processed food, a careful selection of chicken nuggets, ready-meal ravioli, cookies, cereal and the like. For another two weeks, they ate nothing but wholefoods including fruits, vegetables, meat and yogurt.

The participants were offered about twice as much food as they should have needed, and they could eat as much or as little of it as they wished. Crucially, the two diets were controlled so that the snacks and meals on offer contained more or less the same amounts of fat, protein, total carbohydrates, sugar, salt and fibre.

The effect on the volunteers was dramatic. On the ultra-processed diet, people put on weight, gaining an average of 0.9 kilograms. On the wholefoods diet, people lost the same amount on average: just under a kilo. As it turned out, when given free rein to eat as much processed food as they liked, people ate about 500 calories more over the course of a day than they did when choosing from wholefoods.

One possible explanation is that people ate more of the processed food because they found it tastier. On questionnaires, the volunteers rated it as only slightly nicer than the wholefood meals, a difference so small it could have arisen by chance. But how much we eat of any particular food isn't solely down to our conscious opinion of its tastiness.

Ultra-processed food generally comes loaded with just the right amount of fat, salt and sugar for us to find it hard to resist. Think of how common it is to eat more than you intended from a packet of biscuits or crisps. Such food could also have led to overeating in another way, by encouraging people to eat faster. Most of this type of food has water taken out of it during manufacturing – it's crisps, rather than boiled potatoes, say. You end up with a more concentrated form of the food, so with each bite, you are eating more calories.

Another explanation, known as the protein leverage hypothesis, is that a crucial regulator of our appetite is the biological need to eat a certain amount of protein every day. Because meat, a key source of protein, is more expensive than other ingredients, processed foods tend to be low in protein but high in fat and carbohydrates. People whose diets are mainly comprised of processed food end up hungrier because they aren't meeting their protein requirements, so they eat more to assuage that hunger – which, ironically, will end up being more of the low-protein processed fare they are used to eating.

The findings from Hall's study seem to support this last hypothesis, because when people ate the ultra-processed diet, their extra 500 calories a day came mainly from fats and carbs and their protein intake was slightly lower than it was when offered wholefoods.

MOYA STUDIO/ISTOCK

Eating junk food puts us at higher risk of obesity, diabetes and heart disease – but why, exactly?

DEFINING JUNK

The NOVA system classifies each foodstuff into one of four groups according to how much industrial processing has been applied to it.

GROUP 1:
UNPROCESSED AND MINIMALLY PROCESSED FOODS

Edible parts of plants, animals or fungi, such as meat, fruit or mushrooms.

GROUP 2:
PROCESSED CULINARY INGREDIENTS

Substances including oils, butter, sugar and salt that are derived from group 1 foods through processes such as refining, grinding or drying. They aren't normally consumed by themselves, instead being used in combination with group 1 foods.

GROUP 3:
PROCESSED FOODS

Foods made by adding ingredients from group 2 to items from group 1. This includes cheese, canned fish, bottled vegetables and freshly made breads.

GROUP 4:
ULTRA-PROCESSED FOODS

Foods made from base ingredients, such as lactose, oil, whey and gluten, that may have been extracted from foods but have usually undergone modern processing to create things like hydrogenated oils, hydrolysed proteins and high-fructose corn syrup. This is also foods containing additives such as emulsifiers, colourings and flavour enhancers. Final products include many ready meals, cereals, mass-produced breads and fries.

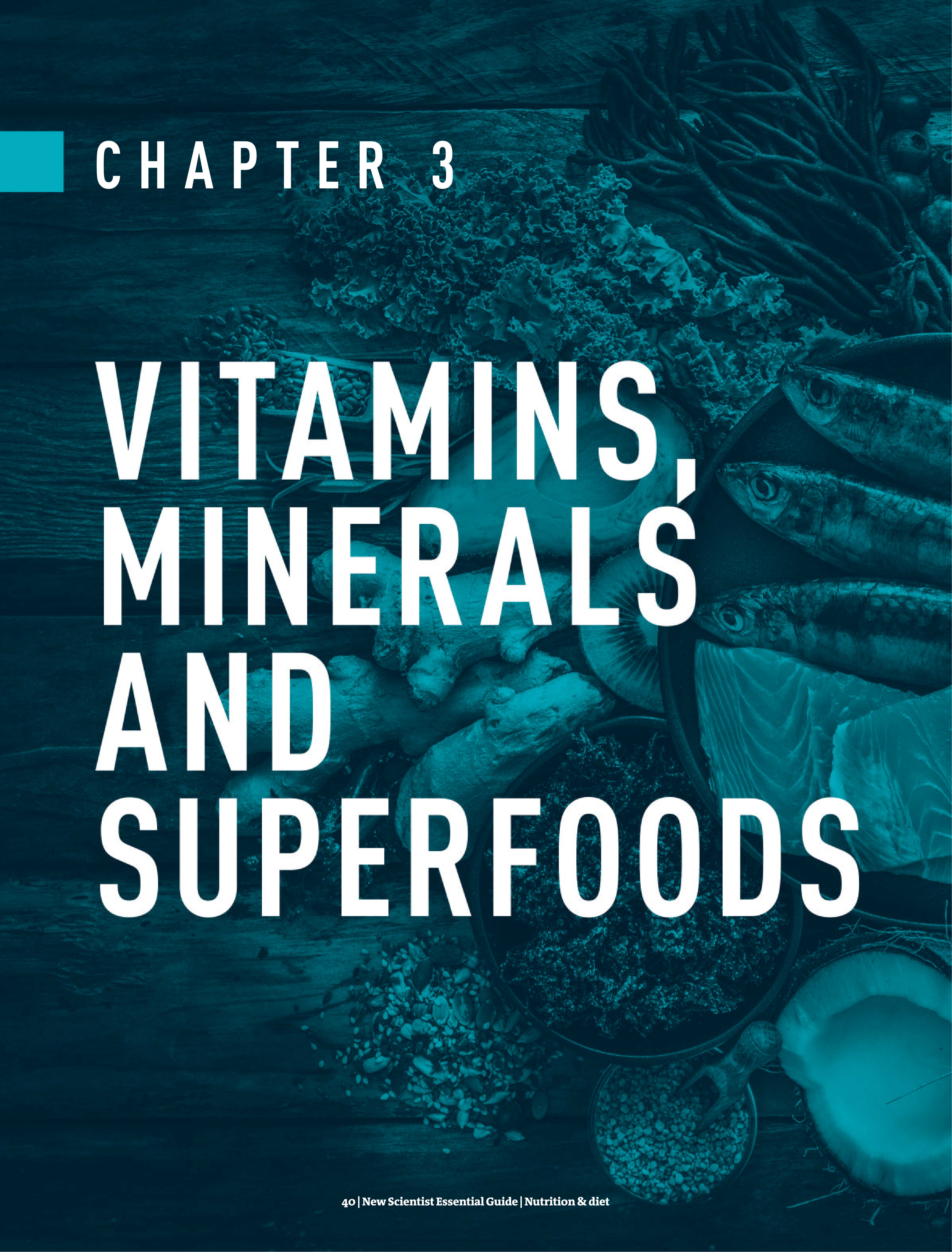
One other theory is that the artificial additives that processed food contains, such as sweeteners and the emulsifiers that help fat-soluble substances mix evenly with water, disturb our gut bacteria. Studies in rodents suggest that both of these chemicals change the ecosystem of the microbiome and that emulsifiers let bacteria encroach on the gut wall, causing inflammation.

←
Turn back to page 16 for more on the microbiome

Any or all of these factors could explain Hall's results – or there could be something else going on. More research is, as ever, needed. Nevertheless, Hall's results have already stimulated action. Soon after the study came out, the UN's Food and Agriculture Organization published a report calling the trial a "solid link" between ultra-processed foods and ill health, and calling for a raft of measures to reduce their consumption, including food labelling, advertising bans and junk-food taxes on such products at the point of sale.

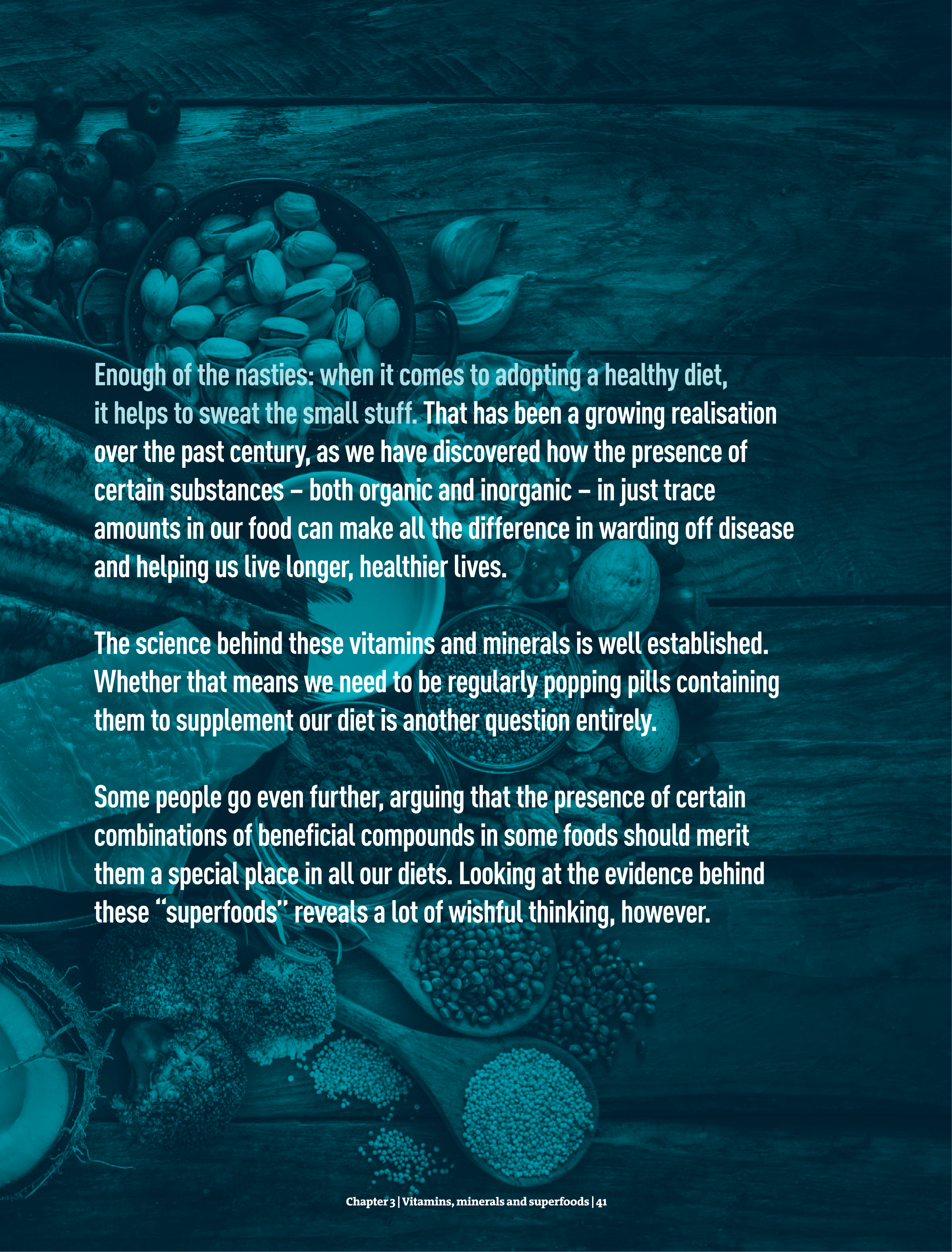
Some campaigners argue that governments should go further still and introduce higher taxes on such foods. This is controversial, though, because processed foods make up such a large part of most people's diet in high-income countries. Processed foods are often cheaper than home-cooked meals thanks to economies of scale and are quicker to prepare, all of which matters if you are working long hours to support a family on a low income.

All things considered, while we may not yet fully understand the mechanisms, the evidence is accumulating that it is probably best to avoid eating too much ultra-processed food. While we wait for the science to catch up, the take-home message is simple: eat wholefoods as much as your budget and circumstances allow. Your body may thank you for it. ▮



CHAPTER 3

VITAMINS, MINERALS AND SUPERFOODS



Enough of the nasties: when it comes to adopting a healthy diet, it helps to sweat the small stuff. That has been a growing realisation over the past century, as we have discovered how the presence of certain substances – both organic and inorganic – in just trace amounts in our food can make all the difference in warding off disease and helping us live longer, healthier lives.

The science behind these vitamins and minerals is well established. Whether that means we need to be regularly popping pills containing them to supplement our diet is another question entirely.

Some people go even further, arguing that the presence of certain combinations of beneficial compounds in some foods should merit them a special place in all our diets. Looking at the evidence behind these “superfoods” reveals a lot of wishful thinking, however.

VITAL VITAMINS

Getting your vitamins is an essential part of any healthy diet, and vitamin supplements are big business. To cut to the chase: there is very little evidence that taking most of them supplies significant health benefits. What is important is to eat a healthy, balanced diet in the first place – which is where it is essential to know your vitamin ABCs.

IN 1911, Polish chemist Casimir Funk made one of the most influential biomedical discoveries of all time. He learned that a disease called beriberi affected those who ate a diet of mainly white rice, but not those who ate mostly brown rice. He isolated a chemical from rice bran, showed it could prevent beriberi and called it "vitamine".

This compound, the organic molecule thiamine, now goes by the name vitamin B_1. It is one of many organic molecules that are essential nutrients the human body can't produce in sufficient quantities and that we must obtain from food. Casimir's breakthrough led to similar discoveries, including the compounds that prevent scurvy and rickets. In 1920, the British chemist Jack Cecil Drummond proposed dropping the "e" and using the umbrella term "vitamin". Vitamins now come in six varieties – some with many sub-varieties, and each with different but essential roles in maintaining healthy vital functions.

VITAMIN A

"Vitamin A" is an umbrella term for several compounds, including retinol and retinoic acid, that we get from our diet. It is vital for growth, vision and development. Dairy products and fish oils are rich sources of it. Provitamin forms, such as the orange pigment beta carotene, get converted into vitamin A in the body. Just one small carrot meets your daily need for 0.6 to 0.7 milligrams.

Yet some supplements provide higher doses, and we store the surplus away in fat, where it can build up to harmful levels. An average daily intake of more than 1.5 milligrams of vitamin A may interfere with the beneficial effects of vitamin D and weaken bones too.

Vitamin A deficiency is the main cause of preventable blindness in children who are persistently malnourished. In well-fed populations, however, several well-designed trials have failed to show any benefit from taking extra, and in fact suggest that supplements may increase the risk of cancer and death.

NITO100/ISTOCK
PREVIOUS PAGE: FCAFOTODIGITAL/ISTOCK

B VITAMINS

There are a total of eight B vitamins essential to the process of turning food into energy (see "Know your B vitamins", right). A lack of B vitamins is related to serious diseases: vitamin B_1 deficiency, for example, causes beriberi, while too little vitamin B_3 leads to pellagra, the symptoms of which include diarrhoea, skin lesions and dementia.

Vitamins B_1, B_2, B_3 and B_9 are often added to fortified flour, and well-nourished people can usually easily get all the B vitamins they need from their diet. Vegetarians and vegans are the exception: vitamin B_{12} is found naturally only in meat, eggs and dairy products.

Supplements of B vitamins are claimed to help prevent heart disease, stroke and cognitive decline, as they reduce levels of homocysteine, a blood compound that may play a role in these conditions. But randomised trials fail to support the idea that supplements have any benefits for heart or brain conditions.

But a special case must be made for vitamin B_9, also known as folic acid or folate. It is important to the formation of red blood cells, and is also believed to be critical to the production of DNA and RNA. Adults need 200 to 400 micrograms of vitamin B_9 daily; leafy greens, salmon and several types of bean are good sources of it.

During pregnancy, however, the recommended intake rises to between 400 and 600 micrograms per day, or more for people with a family history of fetal abnormalities such as spina bifida, in which there is a gap in the spine. This is due to a defect in the neural tube, a structure that forms in early pregnancy. Taking folic acid supplements before conception and in early pregnancy reduces the risk of neural tube defects by as much as 70 per cent.

KNOW YOUR B VITAMINS

We need to top up our B vitamins each day because we excrete any extra in urine. Here are some of the things they do and the foods you can find them in.

B_1 (THIAMIN)

Sources: whole grains, eggs
Action: aids muscle and nerve function

B_2 (RIBOFLAVIN)

Sources: dairy, eggs, rice
Action: keeps skin, eyes and nervous system healthy

B_3 (NIACIN/NIACINAMIDE)

Sources: liver, fish, whole grains
Action: maintains the nervous and digestive systems

B_5 (PANTOTHENIC ACID)

Sources: tomatoes, potatoes, eggs
Action: needed for normal growth and development

B_6 (PYRIDOXINE)

Sources: fish, liver, potatoes
Action: aids production of haemoglobin

B_7 (BIOTIN)

Sources: peanuts, bananas, liver
Action: helps make hormones

B_9 (FOLIC ACID)

Sources: spinach, asparagus, yeast
Action: critical to the production of DNA

B_{12} (COBALAMIN)

Sources: eggs, fish, meat, dairy
Action: critical to DNA synthesis and red blood cell formation

VITAMIN C

Vitamin C, or ascorbic acid, helps us absorb iron and make collagen, a protein that helps wounds heal. Without enough, you could succumb to the bleeding gums and weeping wounds of scurvy.

Our bodies don't make vitamin C, so we need to consume it. Adults require at least 40 milligrams of vitamin C each day, which is easily achievable by eating foods like peppers, broccoli and strawberries. A single orange covers your daily needs.

What about its famed benefits to colds? A recent review found that taking 200 milligrams of vitamin C every day may reduce the severity and duration of a cold, but it won't prevent it in the first place – at least not unless you are a soldier training in extreme cold, a marathon runner or a competitive skier.

A little extra vitamin C is unlikely to harm you, but large doses may cause diarrhoea, heartburn and other problems. The upper limit of your recommended daily intake is about 2000 milligrams.

VITAMIN D

Vitamin D is actually a group of similar fat-soluble chemicals that, among other things, increase the uptake of essential trace minerals, including calcium, magnesium and phosphorus, in our gut.

It is well known that vitamin D deficiency causes the bone disorder rickets, but it has also been linked to an increased risk of autoimmune diseases and greater susceptibility to viral infections. A recent study found that people with very low levels of vitamin D are at a 57 per cent greater risk of death from all causes than those with the highest levels.

Vitamin D is unique in that we can make our own through sunlight on our skin. In the summer, light-skinned people can get more than enough each day by spending 5 to 10 minutes in the sun wearing minimal clothing. Darker-skinned people need about 30 to 40 minutes. You can also get it from food; oily fish is one of the best sources.

About half of adults in the UK and US are thought to be deficient in vitamin D. Most guidelines agree that young children, pregnant women and elderly people should take supplements – and possibly other adults if they don't get out in the sun much. The recommended daily intake is about 15 micrograms, but experts say up to 100 micrograms per day is probably safe.

Some studies have shown a link between being hospitalised with severe covid-19 and low levels of vitamin D. However, some of the risk factors for covid-19 are the same as those for vitamin D deficiency, which means we should be very cautious about postulating a causative link. A recent review of the evidence came to the conclusion that there was only very low-quality evidence for this, with more research needed.

VITAMIN E

We need vitamin E, another group of fat-soluble compounds known as tocotrienols and tocopherols, to help prevent clots and maintain our immune defences. The daily recommended intake for adults is 3 to 4 milligrams in the UK, but 15 milligrams in the US. Either way, you should get plenty from vegetable oils, nuts, seeds and green vegetables.

Vitamin E deficiency in otherwise healthy people is rare – even getting slightly less than you need doesn't appear to cause harm. Because we store it up in fat, however, it can impact those who have conditions that interfere with the body's ability to digest or absorb fat, such as Crohn's disease or cystic fibrosis. Deficiency can lead to muscle weakness and vision problems, and over prolonged periods it can undermine liver and kidney function.

Because vitamin E is an antioxidant – which can neutralise harmful free radicals that are a by-product of cellular metabolism – it has been suggested that it should help prevent cancer and other diseases associated with free-radical damage. But the evidence doesn't support this.

Indeed, it seems that, in high doses, vitamin E supplements may increase the risk of prostate cancer.

With regard to other conditions, ranging from heart disease to age-related macular degeneration and dementia, there is no evidence of benefits. And despite the popular belief that vitamin E cream can make scar tissue less conspicuous, research doesn't bear this out.

VITAMIN K

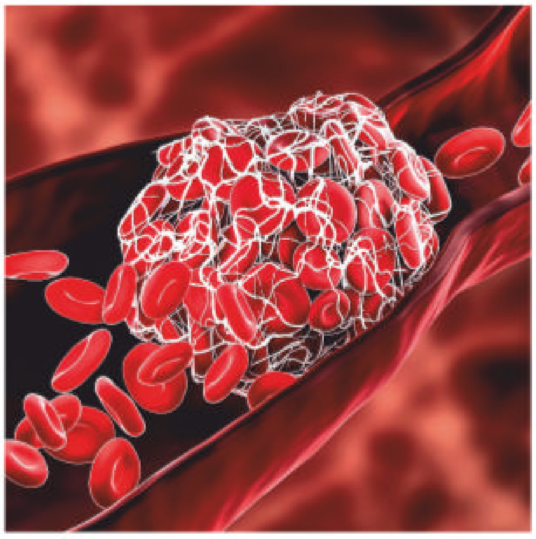
Vitamin K comes in two forms: K_1, found in leafy green vegetables, and K_2, synthesised by bacteria in our gut. It is best known for its major role in blood clotting (its name is derived from its original German title, *Koagulationsvitamin*). The very rare cases of vitamin K deficiency in adults are associated with uncontrolled bleeding. It can interfere with blood-thinning medications, so doctors discourage people who are using these from taking vitamin K supplements.

Depending on body weight, we need around 70 micrograms of vitamin K a day, which we can easily get from a single serving of broccoli. Newborn babies are often deficient, however, so are usually given a booster injection after birth to prevent bleeding problems.

A number of studies have linked low levels of vitamin K with osteoporosis and an increased risk of fractures.

So far, there are only a handful of small studies of vitamin K supplements, and not all point the same way: some suggest extra vitamin K promotes bone health, while others find it makes no difference.

Vitamin K assists blood clotting



LIBRE DE DROIT/ISTOCK LEFT: TIMUR/ISTOCK

MAGIC MINERALS

Vitamins aren't the only nutrients that are essential for healthy bodies and minds. Around 20 inorganic chemical elements, or minerals, are known to be vital for our bodily functions. Here is what we know about some of the most important ones.

CALCIUM

Calcium is important for strong bones and teeth, and its uptake is regulated by vitamin D, making adequate levels of both crucial. We need at least 700 milligrams of calcium each day, and dairy products are a great source of it, as is salmon. People who are lactose intolerant or vegan may struggle to get enough, though. That is worrying: in the long term, too little calcium may lead to bone fractures and osteoporosis.

Bone mass declines after age 35, making you more vulnerable to fractures as you get older. Extra calcium can help: an analysis of 17 clinical trials found that supplements lowered fracture risk by about 24 per cent. Such findings led a US National Institutes of Health consensus panel to recommend in 2001 that older adults use supplements.

Since then, however, many studies have highlighted problems. Too much calcium from supplements can cause kidney stones, and a five-year trial of post-menopausal women found that those taking the supplements were more likely to suffer a heart attack or stroke than those who didn't. A subsequent analysis confirmed the increased risk. In 2013, the US Preventive Services Task Force changed its guidance to recommend against a combination of calcium and vitamin D supplements for post-menopausal women, meaning we are best left getting it from food.

CHROMIUM

Whether or not chromium is a dietary essential is hotly debated. The assumption that we need it is based on unexpected weight loss and other health problems seen in people on long-term intravenous diets lacking in the mineral. For now, scientists presume that we need it and look to how much healthy people get as a guide. This "adequate intake" is 25 to 35 micrograms for adults. For chromium content, broccoli is king, but a diet rich in fruits and vegetables should give you enough.

As to claims that chromium supplementation can burn fat, mediate blood glucose, reduce insulin insensitivity and lower heart-attack risk, there is currently no solid evidence.

IRON

Iron is essential to all life. It is a major component of haemoglobin, the protein that transports oxygen in the blood. It also forms a core part of various proteins involved in metabolism, immune response and the production of neurotransmitters. This makes it critical for basic cellular functioning and brain development, and it also ensures that enough oxygen is delivered to our tissues.

It is relatively easy to get the daily recommended adult intake of 8 milligrams (premenopausal women need at least 15 milligrams), as many common foods contain at least some iron. Meat and fish are good sources, as are beans, dried fruit and tofu.

But some sections of the population are at risk of iron overload. Iron is highly reactive in the body, and easily forms free radicals that will damage cells if its availability isn't tightly regulated. Those at risk include heavy drinkers, people with inflammatory conditions like rheumatism, carriers of certain genetic mutations and those with a long history of taking iron supplements. This may boost their risk of type 2 diabetes and certain cancers. And while studies have shown that boosting iron levels in healthy women whose levels were on the low end of normal reduced their tiredness, we also know that iron overload can also make you tired – making this a mineral to be enjoyed, like most things, in moderation.

MAGNESIUM

Many enzymes require magnesium, including those involved in nerve and muscle function and in regulating blood glucose and blood pressure. A balanced diet should provide the recommended 300 milligrams a day. Nuts and spinach are good sources. But in the US, teenagers and people over 70 often get too little, partly because of a diet high in processed food for the former and poor absorption of the mineral in the latter.

Although a consistently poor diet, diabetes and certain medications can lead to a severe lack of magnesium and symptoms such as heart arrhythmia, it is rare for deficiency to cause illness. The evidence is sketchy on the benefits of supplements, beyond treating such extremes. And despite well-rehearsed claims, there is little clear evidence of any benefit against diabetes from taking magnesium. Some research suggests a protective effect for stroke and heart disease, but when it comes to other health claims for magnesium, including lowering blood pressure, studies show a very small impact, if any. Finally, for treating migraine, there is no clear consensus for or against.

PHOSPHORUS

Phosphorus is a component of bones, teeth, DNA and RNA, making up between 1 and 1.5 per cent of the average human's fat-free mass, mainly in bones and teeth. Like calcium, its uptake is regulated by vitamin D. Because it is so widely present in organic tissues, however, it is also present in most foods we eat, making phosphorus deficiency, or the need for phosphorus supplementation, extremely rare.

POTASSIUM

Potassium works in tandem with sodium to regulate the volume of blood in our body, and helps us get rid of excess salt – and with it, excess fluids – through urine.

High blood pressure is a major cause of heart disease. And the popularity of processed foods, which tend to be high in salt and low in potassium, adds to the problem. In healthy people, the ratio of sodium to potassium should be roughly 1:1. So how do we get the 3.5 grams per day recommended by the World Health Organization? For most of us, this is unsurprisingly down to eating fresh fruit and vegetables.

Still, supplements are popular – sales are worth close to $90 million a year in the US. The pills are occasionally prescribed for people who have trouble absorbing potassium naturally. In otherwise healthy people, potassium deficiency, characterised by fatigue and muscle weakness, is very rare. Supplements may pose a risk for people with high blood pressure or certain kinds of kidney disease that mean they can't get rid of potassium, but are otherwise unlikely to cause harm.

SELENIUM

Selenium is a trace element found in fish, meat, grains and dairy products. It is crucial to the formation of selenoproteins, which are vital for DNA production. In the West, it is very rare for people to lack selenium; an adult only needs about 60 micrograms per day, which a portion of fish or a single Brazil nut can supply.

Selenium can be poisonous in large doses, but the most prominent case of toxicity involved people taking supplements containing more than 200 times the recommended daily intake. Most supplements contain 50 to 200 micrograms. It is claimed that they can help prevent illnesses like cardiovascular disease and cancer, and also stave off age-related cognitive decline. Research into whether selenium supplements could lower the risk of some cancers has been contradictory, leading the US Food and Drug Administration to deem the evidence inconclusive.

Selenoproteins reduce inflammation and stop blood platelets clumping together, so selenium could be beneficial against cardiovascular disease. But again, the evidence is mixed. A review of 12 trials didn't support this claim, and some trials even suggest high selenium intake could increase risk. The results are equally opaque for thyroid disease, and when it comes to preventing cognitive decline, promising findings have been muddled by poorly controlled experiments.

Does sulphur give hair extra bounce?

SULPHUR

Our bodies need sulphur to make proteins, but it is unclear how much we need for health. Supplements containing MSM (methylsulphonylmethane), a sulphur-containing compound found in meat, vegetables and other foods, have been touted as maintaining healthy hair, nails, bowels, metabolism and immune function, as well as warding off cancer. As yet, there is little evidence that supplementing a normal balanced diet brings any benefits.

ZINC

Oyster lovers, rejoice! Zinc plays a vital role in cell division, is required for about 100 enzymes to work and helps with metabolism, wound healing and the production of DNA – and oysters provide more per serving than any other food.

The daily recommended amount is 4 to 11 milligrams. Too little can result in a weakened immune system, hair loss and mental slowness. Some evidence suggests zinc supplements can slow age-related macular degeneration and perhaps also help with symptoms of sickle-cell anaemia, but more research is needed.

The role of supplements is clearer when it comes to colds. A recent review found that doses of 75 milligrams a day or more, if taken within a day of getting sick, shortened the length of a cold. There is no evidence that supports taking extra zinc routinely, however.

THE IMPORTANCE OF OMEGA-3

Besides vitamins and minerals, some fatty acids derived from what we eat are known to be essential for good health. Chief among them are so-called omega-3 fatty acids, such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). These act as building blocks for nerve and brain tissue, and keep our hearts beating regularly and our brains firing.

Without these nutrients, our bodies can't make the hormones that control artery expansion and blood clotting. If you are habitually consuming negligible amounts of omega-3s, it can affect cognitive function. In childhood, low omega-3 levels can be associated with attention deficit disorder and issues like hyperactivity, anxiety and temper tantrums. Omega-3s have been found to help with sustained attention in adults too. Eating high levels of omega-3s has been linked with a reduced risk of cardiovascular disease and even depression.

The vital role of DHA in forming the brain and nervous system in embryos and infants is undisputed, but recent studies find that elderly people may also benefit from increased omega-3 consumption: those with higher levels of EPA and DHA in the blood tend to have greater brain volume, particularly in areas associated with memory.

Our main dietary source of omega-3s, including EPA and DHA, is fish oil. While these substances are only made naturally by marine algae and diatoms, they build up in fish that eat the algae (or that eat smaller fish that do so).

The alternative is fish-oil capsules. These do have their critics – some studies have found that the evidence of benefits is limited. But the bulk of the evidence points to positive effects on heart health – 1 gram of fish oil per day can cut the risk of death by heart attack by as much 20 per cent. Health authorities around the world recommend a minimum intake of 250 milligrams each day, and set no upper limit on daily intake, making these some of the few supplements that do have some solid science behind them.

SOUTH AGENCY/ISTOCK PREVIOUS PAGE: JOKMEDIA/FLOORTJE/ISTOCK

THE LURE OF SUPERFOODS

Foods purported to provide extraordinary health benefits because of the nutrients they contain are big business. We are clearly swallowing the hype – but do any of the claims stack up?

Goji berries supposedly boost immunity and protect against heart disease and cancer

ANDRII POHRANYCHNYI/ISTOCK

YOU don't have to shop at Whole Foods or hang on Gwyneth Paltrow's every nutritional word to have heard the promise of superfoods. In a survey of more than 1000 UK adults in 2015, 61 per cent admitted to buying a food because they considered it a superfood. That is despite the word having no scientifically agreed meaning. It is a term that was invented to sell products – a marketing gimmick.

The European Union has banned the word on packaging unless it refers to a specific claim based on convincing research from an approved list. There are no such regulations in the US, but the Food and Drug Administration can take action if they find any claims to be false or misleading.

Even when claims aren't directly misleading, scratch the surface of the scientific jargon designed to attract health-conscious consumers and things quickly get murky. Studies often show that concentrated extracts or isolated compounds found in abundance in certain foods have particular effects in a Petri dish or a mouse. But that doesn't mean they work in the same way when people eat them. For the average punter, it can all get a bit confusing. Often, they are no better for us than ordinary, everyday fruit and veg. Let's look at how the claims made for a selection of superfoods stack up.

CHIA SEEDS

The ancient Maya used chia seeds for thousands of years to make everything from flour for tortillas to tea. Now they are one of the trendiest superfoods, often said to be a great source of omega-3s (see page 48).

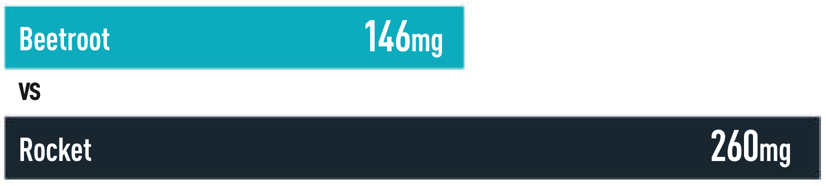
Chia seeds contain roughly 17 grams of omega-3s per 100-gram serving. At first glance, compared with oily fish, that looks impressive: a 100-gram portion of farmed Atlantic salmon gives you 2.2 grams of omega-3s. Unlike salmon, however, the fatty acids in chia seeds come as alpha linolenic acid (ALA), which the body has to convert to eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) to get the cardiovascular health benefits.

We don't do this conversion very well. It varies, but the median efficiency is 10 per cent, so the amount of EPA and DHA you get from 100 grams of chia seeds drops to 1.7 grams, fewer than from salmon. To convert ALA, your body also has to digest the seeds to extract the fats – and we all know that some seeds pass straight through.

So for omega-3s, it might be better to smash up your chia seeds in a smoothie. Then again, if you are after soluble fibre to mop up bad cholesterol, whole seeds are the way to go. Two tablespoons provide around 30 per cent of your daily intake. With chia seeds, you can have your fibre and eat your omega-3s – just not necessarily in the same serving.

When it comes to delivering the active ingredients thought to do you good, how do so-called superfoods stack up against ordinary fare? We compare 100-gram portions.

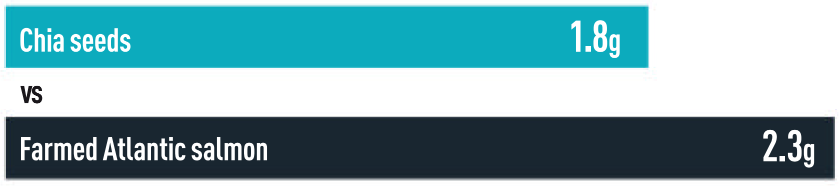
For nitrates, which can help lower blood pressure...



SOURCE: BRITISH JOURNAL OF CLINICAL PHARMACOLOGY, VOL 75, P 677

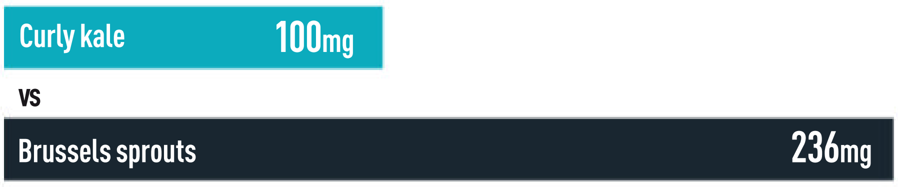
For Omega-3 fatty acids, good for a healthy heart...

This comparison is based on the amount of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) your body gets



SOURCE: USDA SR28

For glucosinolates, which may lower the risk of cancer...



SOURCE: BRITISH JOURNAL OF NUTRITION, VOL 90, P 687

GOJI BERRIES

Long revered in traditional Chinese medicine, goji berries (*Lycium barbarum* and *Lycium chinense*) are said to pep up your immunity, boost libido and protect against heart disease and cancer. But there is precious little research identifying their supposedly unique active ingredients, never mind measuring their health benefits.

What research there is tends to focus on a group of chemicals called Lycium barbarum polysaccharides, or LBPs. According to a review of in vitro and mouse research back in 2015, they can stop tumour growth, regulate blood sugar, protect eye and liver cells from free-radical damage and improve sperm motility.

There are reasons to be sceptical. First, few studies define what LBPs are. Polysaccharides are long-chain carbohydrate molecules, but in the goji berry literature, LBPs appear to include carbohydrate chains linked to proteins – collections that aren't typically considered polysaccharides. What's more, there are no rigorous human trials measuring which components get into the bloodstream and what they do. Even if the studies of LBP extracts turn out to be reliable, their relevance to human nutrition is far from clear.

The other big claim is that goji berries contain high levels of zeaxanthin, a compound linked to the prevention of age-related degeneration in eye cells. But if it is zeaxanthin you are after, you can get your fill from leafy vegetables such as spinach and cabbage or from yellow peppers. And although goji berries do contain more vitamin C than blueberries, you get roughly the same amount from strawberries or lemons.

KALE

Kale's main selling point is a family of sulphur-containing plant chemicals called glucosinolates that give all dark-green vegetables their characteristic bitter taste.

Glucosinolates are broken down in the gut to release glucose and isothiocyanates, which have been shown to stimulate enzymes whose job it is to eliminate cancer-causing chemicals. This much has been shown in animal research, and it is supported by studies linking higher consumption of glucosinolate-rich brassicas to a lowered risk of cancer in humans, particularly that of the gut and lungs.

Given that all brassicas contain similar stuff in similar amounts, however, kale is no better than white cabbage or Brussels sprouts. One analysis revealed that several varieties of cabbage contain as many glucosinolates as kale. What's more, there are more than 100 different glucosinolates, and each gets broken down into a different isothiocyanate, so it is probably best to munch all kinds of brassicas. Be warned, though: boiling reduces glucosinolate content, so the crunchier the better.

KIMCHI & KEFIR

Kimchi is a Korean side dish made of fermented cabbage; kefir is a fermented milk drink from the Caucasus mountains in eastern Europe. These and other fermented foods have been around for centuries. In recent years, however, they have received sudden acclaim as microbiome-balancing paragons of dietary virtue.

Some of their powers are put down to the fermentation process, in which bacteria partially digest the food, releasing a greater hit of nutrients. Indeed, it seems to improve the availability of iron, which is essential for making red blood cells: one recent study in humans showed greater absorption of iron from fermented mixed vegetables than fresh ones.

The biggest health claim, however, is the supposed effects on your gut microbiome – the billions of bacteria that reside in your intestines, quietly regulating all kinds of bodily functions. The idea is that the fermentation process increases the numbers of beneficial bacteria naturally present in the food and, when you eat it, in your body.

→
Find out what else your microbiome is doing on page 16

Animal studies suggest that fermented foods might encourage a healthy mix of microbes in the gut. Adding kefir to the diets of mice, for instance, increased the population of beneficial bacteria like *Lactobacillus* and *Bifidobacterium* in their intestines and reduced potentially harmful ones. In humans, however, caution is needed before we make sweeping health claims. Fermented foods do look as if they could benefit gut flora – but what this might do for your health isn't yet clear.

QUINOA

A handful of human studies have shown that replacing cereals with quinoa, a seed that is cooked like a grain, can reduce blood cholesterol and help people lose weight. But the sample sizes are so small that it is difficult to draw firm conclusions.

Such benefits are typically attributed to chemicals called saponins, thought to work by altering the permeability of the gut. It may also be because they produce a bitter taste that puts people off eating too much. Either way, washing your quinoa before eating it, as many people do, removes the saponins and thus the benefits they might bestow.

SUPERDRINKS

When it comes to hydration, what could be better than water? Several things, if recent claims of special health benefits are to be believed.

BEETROOT JUICE

No longer just soaked in vinegar and served from a jar, beetroot is now lauded as a blood-pressure lowering, metabolism-revving superfood. Its main do-gooders are said to be nitrates. These are converted into nitrites by saliva and then pass through the stomach, where they are converted into nitric oxides – compounds that relax blood vessels, bringing all sorts of cardiovascular benefits.

Studies have shown that dietary nitrate brings down blood pressure and improves circulation. One study even found that a 500-millilitre glug of beetroot juice per day, containing 400 milligrams of nitrate, improves exercise performance, buying people an extra 90 seconds of intense exercise before exhaustion.

Maybe don't overdo it, though. The European Food Safety Authority puts the safe limit for dietary nitrate intake at around 260 milligrams per day for an average adult – that is equivalent to two whole beets or one 300-millilitre glass of beetroot juice. The main concern is that if nitrites combine with protein in the stomach, they could potentially form nitrosamines, which may contribute to gastric cancers.

WHEATGRASS

Fans of the wheatgrass shot insist that it will flood your tissues with oxygen. The story goes that chlorophyll, the compound that plants use to make sugars via photosynthesis, is structurally similar to haemoglobin, the protein in red blood cells that carries oxygen around the body. So as there is more chlorophyll in wheatgrass shoots than in other edible plants, you get more oxygen.

That's nonsense. Chlorophyll is found in similar concentrations in many green vegetables. More to the point, there is no evidence to support the idea that chlorophyll functions anything like haemoglobin. Even if it did, it wouldn't get into the bloodstream because chlorophyll gets broken down in the gut.

COCONUT WATER

This clear liquid tapped from young, green coconuts has been dubbed "nature's sports drink". But studies comparing it with isotonic sports drinks and ordinary water found no difference in terms of how well they hydrated volunteers after vigorous exercise.

Claims for coconut water appear to rest on the idea that its higher potassium levels enhance water absorption. That doesn't stand up: according to another study, neither coconut water nor a potassium-rich sports drink score higher on fluid retention than water.

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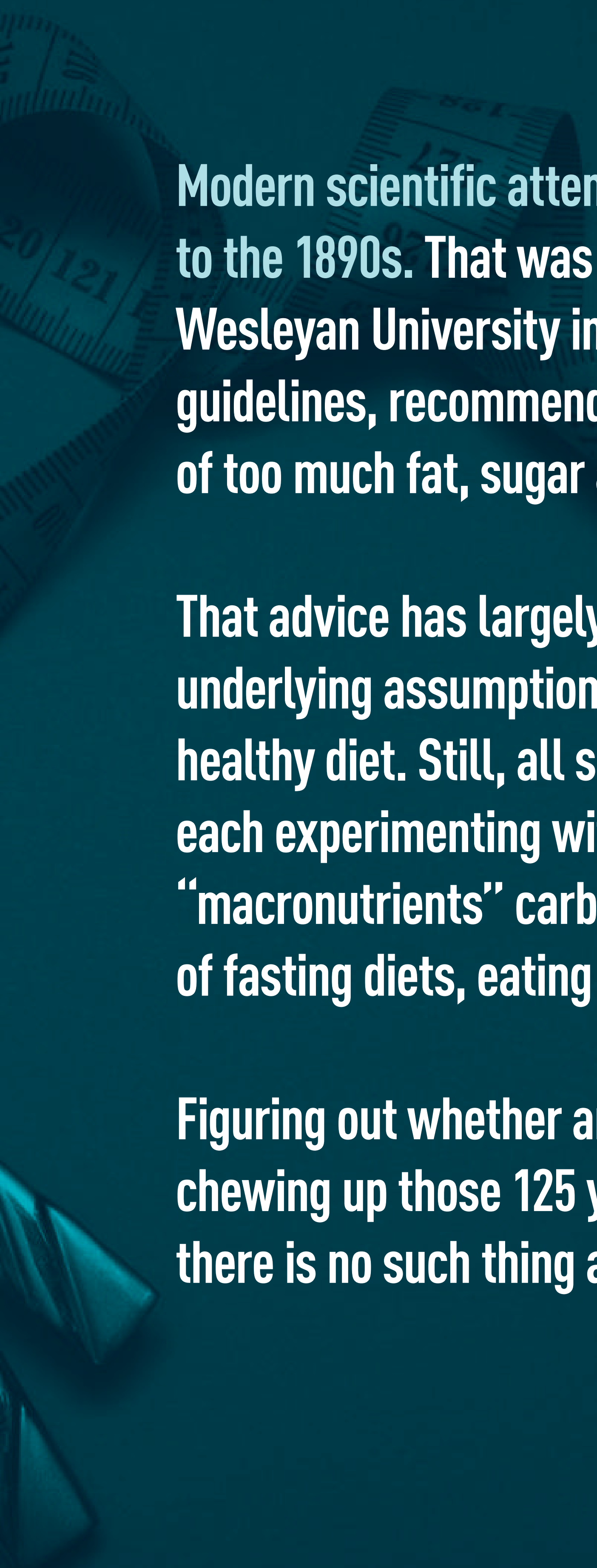
CHAPTER 4

DIETING AND WEIGHT LOSS



Modern scientific attempts to define a healthy diet date back to the 1890s. That was when nutrition pioneer Wilbur Atwater at Wesleyan University in Connecticut published the first ever dietary guidelines, recommending variety, moderation and the avoidance of too much fat, sugar and starch.

That advice has largely stood the test of time – along with its underlying assumption that there is such a thing as a universal healthy diet. Still, all sorts of food fads have come and gone, each experimenting with eating different balances of the main "macronutrients" carbohydrate, protein and fat – or, in the case of fasting diets, eating none of them at all.

Figuring out whether any of them work brings us closer to chewing up those 125 years of orthodoxy. In fact, it may be that there is no such thing as one healthy diet.

CUTTING CARBS

One food fad seems to stick around. Whether you call it low-carbing, Atkins, keto or paleo, the principle is the same: cutting down on starchy food and filling up on fat and protein. But are low-carb regimes an easy way to diet or a recipe for heart attack?

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PREVIOUS PAGE: TAFIPHOTO/ISTOCK

LOW-CARB diets first became popular in the 1970s through New York cardiologist Robert Atkins, who lost weight himself this way and recommended it in diet and cookery books. His advice to fill up on steaks, cream and butter, while shunning most fruit and vegetables, made him a medical pariah. Critics said people wouldn't be able to stick to it, and if they did, it would kill them, says Eric Westman, an obesity specialist at Duke University in North Carolina, who studied under Atkins.

By the early 1990s, however, randomised trials were showing that such diets are at least as good as low-fat ones for weight loss, often a little better. In one trial, people on a low-carb diet lost an average of 4.4 per cent of their body weight after a year, compared with 2.5 per cent among those in a low-fat group. And contrary to the warnings, people's cholesterol levels and results from other blood tests generally moved in the right direction. "That was a big moment," says Westman, who led some of those studies.

The central idea behind low-carb diets is that weight control requires more than just calorie counting. For a start, the same number of calories from fat or protein makes people feel fuller than if they come from carbs, which explains why people on low-carb diets report

less hunger than those on low-fat ones. Westman's studies helped show that, although most people on low-carb diets don't calorie count, they tend to consume just 1200 to 1500 calories a day, much less than the recommended 2000 calories per day for women and 2500 for men. They eat less because they aren't as hungry.

Usually, our cells are fuelled by glucose, a simple sugar that all other forms of sugar or starch are converted into. Glucose is highly reactive, so our bodies normally keep the amount of it in our blood within a narrow range to avoid damage to blood vessels and cell structures. When glucose levels rise after eating, we quickly release the hormone insulin, which instructs cells to start taking up glucose, using and storing it.

Insulin has a host of other effects, but they can be summarised as signalling to our bodies that we have had an influx of calories and we need to stash them away. Crucially, insulin makes fat cells turn glucose into fat and store it. But in the absence of glucose, the body has an alternative fuel source: fat. Depending on the cell type, stored fat may be turned into fatty acids or into molecules called ketones, which can be used for energy. This normally happens a little overnight, when we go for several hours without eating any carbohydrates.

The raison d'etre of low-carbing is to minimise insulin release and to be fuelled as much as possible by ketones. For most people, a shift into what is known as ketosis happens within a few days of dramatically cutting carbs. Eating very low levels of carbs is also known as a ketogenic diet.

As well as people trying to lose weight, many others have adopted low-carbing or the keto diet because they see it as a way of living healthily and prolonging lifespan. Some adherents believe entering ketosis has a range of metabolic benefits, including warding off cancer and Alzheimer's disease, although there isn't good evidence for this.

What has recently granted low-carbing more legitimacy, though, is its effects on type 2 diabetes. This condition occurs when the body's cells become less sensitive to insulin – a state known as insulin resistance – which leads to dangerous rises in blood sugar after meals. Long term, these sugar surges contribute to the many health consequences of diabetes, including nerve damage and kidney and heart disease.

The medical orthodoxy is that, because diabetes raises the risk of heart disease, it is even more important that those affected avoid saturated fats, found mainly in red meat and dairy products, because these are thought to raise blood cholesterol and lead to blocked arteries. The UK National Health Service advice for type 2 diabetes is that people should keep fat to a minimum and eat starchy foods like pasta, for instance.

Yet this ignores the fact that people with diabetes may see two benefits from low-carbing. As well as weight loss improving their insulin sensitivity, avoiding starch and sugar reduces those harmful blood sugar spikes. Starch is basically long chains of sugar molecules. People with diabetes often measure their blood sugar at home, and can see for themselves that starting the day with bacon and eggs gives less of a sugar surge than toast or cereal do.

> “Many people now see low-carb diets as a way of being healthy and prolonging lifespan”

Sceptics might say that while a few days of low-carbing leads to lower blood sugar levels, it is hard to stick to this way of eating. There is mixed evidence on the issue. For instance, a review of 10 randomised trials found that low-carb diets were more effective than low-fat ones at improving blood sugar control in people with diabetes over the first year, but the differences disappeared after that. But there is evidence that, for those who keep it up, the health benefits can be longer term, such as a study of 128 people with type 2 diabetes who went to low-carb counselling sessions run by David Unwin, a family doctor in Southport, UK. After an average follow-up of two years, about half had been able to stop taking all their diabetes drugs.

Because of results like these, diabetes doctors and patient support groups have started questioning the low-fat orthodoxy too. Bodies such as Diabetes UK and Diabetes Australia now say that low-carbing is a valid option for weight loss. Ten years ago, that would have been unheard of. The American Diabetes Association went a step further in 2020 and said that out of all dietary strategies, low-carbing has the most supporting evidence for improving blood sugar control.

Yet, as more people have adopted this way of eating, there have been renewed questions over its safety. In some cases, low-carb diets can lead to an alarming change in levels of “bad” LDL cholesterol. Although these seem to be in the minority, the number of these “hyper-responders”, as they are coming to be called, is unclear. In one of Westman’s trials from 2004, two people out of 59 randomly assigned to a low-carb diet dropped out because their bad cholesterol levels rose.

Westman and others say they most often learn of this response in people who are slim and relatively muscular. He believes he may now be hearing about more of these cases because low-carbing is increasingly being adopted as a longer-term approach to healthy eating, not simply a short-term strategy for weight loss.

You don’t have to be slim and muscular to be a hyper-responder, though. Vipan Bhardwaj, a family doctor in Wokingham, UK, saw bad cholesterol rise in two out of 38 of his patients who began low-carbing for diabetes. “It scared the bejesus out of us,” says Bhardwaj.

What is strange about hyper-responders is that while their LDL level goes up, their other health markers tend to move in the right direction. These include their HDL, triglycerides, blood pressure and several other measures linked with glucose and insulin response. This was true of Bhardwaj’s hyper-responders. He got further reassurance by scanning the arteries to their heart to check for any plaques. “You see what’s really going on underneath the bonnet,” he says. “They were absolutely fine.”

The fact that some people see a rise in their bad cholesterol levels on a low-carb diet while other measures improve is now leading some to question whether LDL really is a key determinant of the risk to our hearts. Much of the case against this form of cholesterol has been built on population studies done in the past few decades, which found that people with higher LDL levels were more likely to have heart attacks. ›

←
Turn back to page 24 for more on saturated fat and cholesterol

But these kinds of studies can only find correlations between blood markers and health outcomes, not prove that one leads to the other. It could be that something else is the root cause of heart disease, which increases LDL levels as a side effect.

The chief suspect for that something else is insulin resistance. This idea isn't drastically at odds with our current understanding of the root causes of heart disease. Doctors already recognise that type 2 diabetes, obesity and heart disease frequently co-occur – so much so that the triad has its own name, "metabolic syndrome". Yet, rather than heart disease being a disorder of "faulty plumbing" in which our arteries get blocked up because we consume too much fat, the real problem could be a predisposition towards insulin resistance, which in turn promotes higher insulin levels, fat storage and heart disease. "If that is true, the medical research community made a terrible mistake, and we've yet to fix it," says Gary Taubes, a US science journalist, who has long been a proponent of low-carbing and has written a book called *The Case for Keto*.

Another important strand of evidence that made us believe LDL is important was the success of LDL-lowering drugs called statins, which reduce heart attack rates, according to multiple randomised trials. But several other drugs that lower LDL levels don't protect against heart attacks, and statins have many effects on the body, including dampening low-level systemic inflammation. It may be that statins actually protect the heart through their anti-inflammatory effects.

While practising physicians focus on their patients' LDL levels, these days, cardiologists who study biomarkers of heart health debate whether LDL levels really are the most important indicator, or whether things like the ratio of total cholesterol to HDL, or that of triglycerides to HDL, would be more useful. Hyper-responders would be fine if judged by either of those

Statins (above) are routinely prescribed against heart disease. But might too much starchy food like pasta be part of the problem?

“One trial has been interpreted as evidence both for and against low-carb diets”

WHAT ARE CARBOHYDRATES?

Carbohydrates are one of the three main constituents of the food we eat, along with fat and protein. Carbohydrates can be further divided into sugars, starches and fibre, also known as roughage. Chemically, sugars are small, simple molecules, such as glucose and fructose. Starches are built from chains of sugar molecules strung together, while indigestible fibre is made of more complex, cross-linked chains.

Standard dietary guidelines generally advise that we try to get most of our energy from starch, by eating foods such as potatoes, bread, pasta and rice. The thinking is that fat makes us put on weight as it has more calories, and saturated fats found in meat and dairy are particularly bad for us as they increase blood cholesterol levels, which blocks our arteries and causes heart attacks. Advocates of low-carb diets beg to differ.

UGURHAN/ISTOCK TOP: GANNET77/ISTOCK

metrics: generally their HDL levels rise and their triglyceride levels fall.

One trial often used to underscore the dangers of low-carb diets found that people with type 2 diabetes saw their LDL levels rise by about 10 per cent on average after a year on the regimen. However, there was improvement in most of the other 25 health markers tracked, such as weight, blood pressure and HDL. Participants’ overall risk score for heart disease fell by 12 per cent.

As an illustration of how confusing this is for the public, this particular trial is cited both as evidence against low-carbing – because LDL went up – and in favour of it, because overall risk went down.

So where does the uncertainty leave the average person who wants to lose a few pounds? The emergence of hyper-responders shouldn’t stop people from trying low-carbing, says Westman. “That would be crazy, like telling someone not to take a useful drug because it sometimes has a side effect.”

Tracy Parker, a dietitian for the British Heart Foundation, says that if people are determined to reduce their carb intake, the safest bet is to replace carbohydrates with oils from plants and fish. However, she admits this would make what is already a restrictive diet even more so, because people would have to avoid not only all starchy and sugary foods, but also meat and dairy products.

Of course, low-carbing isn’t the only way to lose weight, and obesity clinics often provide several different weight loss options. The idea that different people might do better on different foods is starting to gain more traction. In fact, read on to find out why there is no such thing as a single healthy diet that works for everyone. Instead, our individual genetics, habits and gut microbiomes may all influence how our bodies deal with the nutrients in our diet. ▮

Protein is in vogue. From high-protein bread, cereals and noodles to protein-enhanced cheese and yoghurt, and even ice creams and water loaded with the muscle-building macronutrient, the idea that more protein is more beneficial seems to be everywhere. The science says something different.

HOW MUCH PROTEIN DO YOU NEED?

WE HAVE known for a long time that the protein we get from food is vital. Proteins are assembled from a repertoire of 20 amino acids, the basic building blocks of bone, muscle, skin and blood. It is especially important that we get enough of the eight so-called essential amino acids because, unlike the others, our bodies can't make them. In recent years, however, many of us have become convinced we need more of the stuff.

All of which is a bit strange, because pretty much everyone is getting enough protein already. Daily average consumption of protein in the UK is 87 grams for men aged between 19 and 64 and 67 grams for women. This is about 50 per cent more than they require, according to official guidelines. Only people routinely engaging in endurance, speed or strength sports may need more in their diet (see diagram, right).

Contrary to popular perception, there is little evidence that protein-fortified food increases strength, muscle mass or endurance in the general population. The science is less conclusive for people regularly doing resistance training, with some studies leaning either way.

The other big claim is that consuming more protein will help you lose weight: by keeping you feeling full for longer, it stops you from snacking. A 2016 report by Mintel found that a quarter of people who consume high-protein food or drinks in the UK did so to lose or maintain weight.

Here, once again, the science isn't clear-cut. Research has linked the consumption of protein to the release of compounds known as peptides in the gut that are associated with feeling full. However, there is no

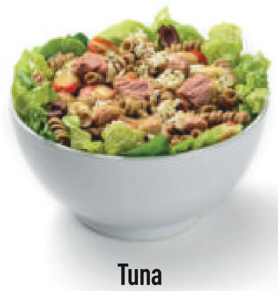
In the UK, adults are advised to eat 0.75g of protein per kilogram of body weight per day. This means men of average weight should eat 56g and women 45g daily. This can be achieved in just a few snacks and meals

MEN

WOMEN

Small glass of semi-skimmed milk (5g protein)

Porridge (11g protein)



Tuna salad (12g)

Cheese sandwich, wholewheat bread (15g)

Bowl of pasta with chicken (39g)

Yogurt and handful of walnuts (16g)

For people regularly lifting weights, the British Nutrition Foundation suggests consuming 1.2-1.7g of protein per kg per day. Taking the upper end of that scale and using the weight of an average person just starting out on a strength-training programme, here is how to get what is required

MALE WEIGHTLIFTER

2 eggs on wholewheat toast (21g protein)

Small glass of semi-skimmed milk (5g)

Cottage cheese on wholewheat toast (20g)

Whole chicken breast and lentils (72g)

Handful of almonds (10g)

TOTAL: 128g

FEMALE WEIGHTLIFTER

Porridge (11g protein)

Small glass of semi-skimmed milk (5g)

Chicken sandwich on wholewheat bread (28g)

Handful of almonds (10g)

Bowl of pasta and grilled salmon (31g)

Yogurt and handful of walnuts (16g)

TOTAL: 101g

SOURCES: BRITISH NUTRITION FOUNDATION. NUTRITION.ORG.UK/NUTRITIONSCIENCE/NUTRIENTS-FOOD-AND-INGREDIENTS/PROTEIN.HTML?START=4 US DEPARTMENT OF AGRICULTURE HTTPS://NDB.NAL.USDA.GOV/NDB/FOODS/

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evidence that feeling fuller in the short term necessarily translates to long-term weight loss, and a 2011 review of eight randomised controlled studies looking at this found there was no conclusive evidence that high-protein diets were better for weight loss than high-fat or high-carbohydrate diets. One factor is that products touting extra protein can sometimes come laden with additional helpings of sugar.

Another thing that rarely gets mentioned is the risk associated with stuffing yourself full of protein. There are good reasons why the UK Department of Health advises adults to avoid consuming more than twice the recommended daily protein intake. When the body has more protein than it needs, the excess is broken down and waste products are filtered from the blood to be excreted as urine.

Research, albeit mostly in animals, has suggested that greater protein intake increases the workload on the kidneys, which do the filtering, potentially leading to damage in the long term. This hasn't been confirmed in human studies and some researchers are sceptical. However, randomised controlled studies have shown that people with kidney disease who are put on low-protein diets are less likely to have renal failure or die from complications linked to their condition than those on normal diets. Excess protein also leads to elevated levels of urea in the blood, and some research has linked consistently high levels to an increased risk of cardiovascular disease.

So despite what you read on supermarket shelves or in fitness magazines, you probably don't need more protein. Indeed, given the way the body deals with the excess, most people who splash out on high-protein water or any other products from the ever-growing protein-fortified range might just as well flush their money down the toilet. ∎

DOES FASTING WORK?

Fasting diets are getting ever more popular, amid promises of weight loss and better health. But can going without food really make you healthier?

FASTING has been part of religious practices around the world for millennia. It has slipped into the consumer mainstream over the past decade, in various forms: take the 5:2 diet, where you fast for two days a week and eat normally for the others; the 16:8, where you eat within an 8-hour window and fast for 16 hours; and alternate-day fasting.

That comes on the back of animal studies that have found that organisms from single-celled yeasts to rodents age more slowly and live longer when their calorie intake falls to 40 per cent of that of a group of animals eating normally. Research in people who are overweight also suggests that skipping meals could have numerous health benefits.

There is growing evidence, too, that periodically going without food puts our bodies into a kind of emergency mode, where they conserve energy, make repairs and prioritise mental clarity to solve the problem of finding food. "If we accept that the Palaeolithic was the environment in which most modern human adaptations were shaped, including dietary ones, the hunter-gatherers then were adapted to periods of feast and famine," says Stanley Ulijaszek, a nutritional anthropologist at the University of Oxford. "This could well be a more natural state for us than 'three meals a day'." As well as weight loss, proponents claim that intermittent fasting could help protect against cancer, diabetes and disorders like Parkinson's and Alzheimer's disease.

Thanks to animal studies, we know a fair amount about what happens in the body when food is scarce. A lack of nutrients kick-starts a process called autophagy, in which cells break down and damaged or dysfunctional parts are recycled and used as fuel. The thinking is that this system probably evolved to

> **“Periodically going without food puts our bodies in a kind of emergency mode”**

maximise the chances of surviving famine.

Autophagy happens at a low level in healthy cells, but becomes less efficient as we age. Sluggish autophagy lets the inside of cells gunk up and has been linked to many age-related diseases, including cancer, and to the ageing process itself. Some researchers believe that the rise in health problems like cancer and type 2 diabetes has a lot to do with the fact that many people no longer go hungry.

Another effect of fasting is that the body starts to run out of glucose in the blood and glycogen stores in the liver, causing ketosis as the liver starts converting fats into ketone bodies for the muscles and brain to use as fuel. This is why fasting almost always causes weight loss of somewhere between 2.5 and 8 per cent.

One crucial question is how long you need to fast to kick-start these processes. Valter Longo, a gerontologist at the University of Southern California in Los Angeles, advocates a radical form of fasting in which you only do it once a month, albeit for five days. According to a study he did in 2017, after three months, people on this fasting-mimicking diet dropped body weight and fat, and ended up with lower blood pressure and lower levels of a hormone called insulin-like growth factor-1 (IGF-1), which is thought to play a role in ageing and disease. They also had lower levels of inflammation markers and cholesterol, among other benefits.

Mark Mattson at Johns Hopkins University in Baltimore, Maryland, thinks you don’t need to deprive yourself of food for that long to see the benefits of fasting. “The liver holds maybe 700 calories-worth of glycogen and people’s general daily activity around the house burns maybe 70 calories an hour,” he says. If you’re not eating during that time, “you do the math, it’s around 10 hours”. Add in exercise and the switch can happen even faster. A vigorous run can burn 100 calories in 10 minutes. Get your sports shoes on in the hours after your last meal and it won’t take long to hit ketosis, says Mattson.

Fasting proponents often claim it also gives them a cognitive boost. So far, though, no controlled studies have been done to investigate the link between fasting and cognition in humans, and the only hints about what might be going on come from mice. Mattson’s group found that switching to ketosis gives the brain a boost, stimulating the release of a chemical called BDNF, which promotes new connections between neurons and stimulates neurons to make more mitochondria, which generate energy.

And as far as autophagy and regeneration is concerned in humans, so far the evidence is purely circumstantial. We don’t know whether any new cells are healthier than what was lost, either, or whether any of this adds up to increased longevity. Epidemiological studies have linked both low and high IGF-1 levels to early death, with high IGF-1 levels linked to increased cancer risk and low IGF-1 to cardiovascular disease.

So does fasting provide any benefits for otherwise healthy people? Michelle Harvie, the researcher at the University of Manchester in the UK who came up with the 5:2 diet, says we just don’t know. “Intermittent dieting is a proven method for weight loss… we don’t know benefits or harms for healthy weight or underweight people.” There is also some concern that intermittent fasting may provide validation for those with eating disorders that invove them rejecting food.

When a person is a healthy weight, their bodily clear-out functions work fine on their own, says Fredrik Karpe at the Oxford Centre for Diabetes, Endocrinology and Metabolism. “Any normal physiological system, in a healthy, lean human being, eating well, exercising, doing what the body likes to do, all these things work. That’s why healthy people who exercise and eat normal things live longer than overweight people.” ▮

SHOULD YOU EAT WHEAT?

A staple food for millennia, and the principal source of complex carbohydrates in many people's diets, wheat is now accused of causing everything from gut ailments to mental health conditions. Our current epidemic of gluten intolerance may say more about our psychology than our physiology, however.

WHEAT was one of the first crops to be domesticated. For thousands of years, it has been a staple food for a large swathe of the world. It is deeply ingrained in our culture and consciousness.

Listen to its detractors today, however, and you will hear that wheat is toxic and addictive, causes food allergies, bloating and lethargy, increases your desire for junk food and makes you fat. Others even blame it for various mental health conditions – and all down to the fact it contains gluten.

Gluten is a composite of two proteins, glutenin and gliadin, and forms a network of fine, stretchy strands when wet. This is what gives dough its elasticity and makes it so versatile, allowing it to be turned into everything from bread to cakes to pasta. Gluten is found in other grains besides wheat, including barley and rye. Going truly gluten-free means excluding the vast number of foods containing these grains, and often things like oats, which are processed with the same machinery as wheat.

All this is entirely necessary if you have a serious condition called coeliac disease. It is caused by the immune system mistakenly producing antibodies against gluten that damage the hair-like villi that line the gut, leading to malnutrition. Blood tests for the antibodies suggest that it affects about one person in

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Many people avoid wheat for its gluten content, but is gluten the real problem?

100. Far rarer are wheat allergies, which can involve one or more of several proteins, not just those in gluten. Symptoms appear rapidly if wheat is eaten or flour dust breathed in, or sometimes if people exercise soon after eating wheat.

What has been on the up in recent years, however, is non-coeliac gluten sensitivity, or NCGS. Peter Gibson at The Alfred Hospital and Monash University in Melbourne, Australia, was first to test its origins in a randomised, placebo-controlled trial. He recruited 34 non-coeliacs with gut symptoms that largely disappeared when they went gluten-free. He kept them on a gluten-free baseline diet, and also gave them bread and muffins that – unbeknown to the participants – either did or didn't contain gluten. Those who ate the gluten-containing muffins reported a worsening in abdominal symptoms and tiredness.

Gibson's hunch, however, was that the problem was caused not by gluten, but by a set of sugars dubbed FODMAPs (fermentable oligosaccharides, disaccharides, monosaccharides and polyols) that are also found in wheat and wheat products. So Gibson did another study with 37 recruits with suspected NCGS. This time, they were put on a diet that was both gluten-free and low in FODMAPs. Then some had high or low amounts of gluten, or a milk-protein control, added to their diets. There was no difference between the three groups' response, suggesting that gluten isn't in fact the root cause of their problems.

FODMAPs are poorly absorbed in the small intestine and so tend to be eaten by bacteria living further down the digestive tract, producing an abundance of gas. They also attract water, leading to bloating, flatulence, loose stools and worse in people with sensitive bowels.

Crucially, FODMAPs aren't just found in wheat, but in foods including many fruits, vegetables and dairy products, so no one is suggesting remaining on a low-FODMAP diet permanently. Some people find they can avoid the problems associated with wheat by simply avoiding cooking with onions, which are very high in fructans, a group of FODMAPs also found in wheat.

The small size of the studies done so far, though, means the science is far from settled. There may be a subset of people who don't have coeliac disease but who do get gut problems when they eat gluten, through an as-yet-undefined mechanism.

There is also evidence to suggest that people who eat food with a low glycaemic index (GI), which release sugars slowly, have a lower risk of developing diabetes than those who eat lots of highly refined, sugar-rich foods. But there is no reason to single out wheat, or other grains, on this basis. The GI of wheat-containing foods varies and is typically lower than that of, say, cooked potatoes.

We also now know that the GI of food is different for different people. What's more, your risk of getting conditions such as diabetes is likely to depend on a complex interplay between your genes, the number and form of calories you consume and how much exercise you do, not solely on whether you eat wheat.

↓
The next section has more on the effects of a high glycaemic index

There remains the claim that wheat is addictive. This is based on the idea that proteins break into fragments that act on opioid receptors in the brain, making us want more. These opioid-like peptides do exist, but whether they affect the brain in any way is unclear. And even if they do, proteins from milk, rice, meat and spinach all produce similar fragments, so again there is no reason to single out wheat.

Similarly, there is no reason to believe wheat is any more fattening than any other carbohydrate. It is just one of the most commonly eaten, so when it is dropped from the diet, it can help you lose weight. Cutting out wheat, after all, means cutting out many high-calorie foods, such as cakes and biscuits.

But swapping such foods for fruits and vegetables is one thing. Swapping wholewheat bread for processed, gluten-free substitutes is quite another. This is bad because not only does eating a high-fibre diet curb your appetite, it also reduces your chances of developing several cancers. And wheat, particularly wholegrain wheat, is a major source of fibre – another thing to bear in mind if you are thinking of cutting it out. ▮

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PERSONALISED NUTRITION

What is good to eat varies so much from person to person that researchers are increasingly arriving at an unexpected realisation: that a universally wholesome diet is a fiction. Instead, the science of nutrition is hot on the heels of a new recipe for healthy eating.

IN 2014, a team at the Weizmann Institute of Science in Israel was probing the effects of artificial sweeteners. Immunologist Eran Elinav and mathematician Eran Segal were specifically interested in whether sweeteners were actually worsening the epidemics of obesity and diabetes that these substances were supposed to help cure. They and their colleagues fed saccharin to healthy human subjects and watched what happened.

One measurement they took was glycaemic response: whether consuming sweeteners caused subjects' blood sugar to rise. This is a normal reaction to eating, but if glucose rises and falls too quickly, or "spikes", it is a marker of poor metabolic health. "People who have regular glucose spikes are more likely to develop diabetes and put on weight than people who don't," says geneticist Tim Spector at King's College London.

People given the same foods are expected to have broadly similar glucose spikes. This is the concept behind the glycaemic index (GI), a measure of how quickly a given foodstuff is converted into glucose and diffuses into the bloodstream. Foods with a high GI – for example sugary foods and drinks and starchy foods such as white bread, potatoes and white rice – are generally to be consumed with moderation.

What the researchers saw took them by surprise. In some people, glucose spiked dramatically, but some had no spike at all and others were somewhere in the middle. In other words, it was a highly personal response.

It sent Elinav and Segal on a voyage of discovery, revisiting the orthodoxy on glycaemic response. In one experiment, they and their colleagues compared industrially produced white bread with artisan, wholegrain sourdough. Based on GI, they expected the white loaf to always generate a bigger glucose spike, but for some people mass-produced white bread was "healthier" than wholegrain sourdough. "We were stunned," says Elinav. "You give people a slice of white bread, some people don't spike at all and others spike to diabetic levels, though on average they spike to exactly the glycaemic index of white bread. And this is true for almost any food."

It was a seminal moment: the paradigm of the one-size-fits-all diet is inherently flawed. If one person's glycaemic response to a given food is the opposite of another's, then the same food can't be good for both people.

This finding tallies with that of another study on 800 volunteers led by Elinav and Segal, which is now widely regarded as the foundational paper in the new field of "precision nutrition". They gathered information on each participant's age, gender, lifestyle and medical history, measured their body mass index and waist-to-hip ratio and took stool samples to reveal people's microbiomes. Then they monitored the volunteers' blood glucose for a week while getting them to exhaustively log what they ate and when, plus their sleep and activity patterns. In total, the researchers recorded glycaemic responses to more than 52,000 meals. As hinted at by their earlier studies, these were hugely individualised, even after eating identical meals.

When they analysed all the data using a machine-learning tool, they found that one of the strongest predictors of an individual's glycaemic response to any given meal was their biometric data, especially microbiome composition. This suggested it should be possible to design a low-GI diet for any individual based on a few measurements.

As proof of that pudding, the team then recruited 26 more volunteers – this time people with prediabetes – then ran them through the volley of tests and designed personalised diets. Everyone got a good diet and a bad diet, each of which they ate for a week while being monitored. As hoped, the good diet significantly improved their glucose responses and the bad one made them worse. Yet, unlike the diets that are routinely recommended for people with prediabetes, some of the good diets contained some pretty unorthodox health foods. "Some people could consume beer or chocolate or ice cream as part of their good diet, but not tomatoes," says Elinav.

Spector's group recently published the results of what he says is "the most intensive nutrition intervention study that's been done". PREDICT-1 – the Personalized Responses to Dietary Composition Trial – recruited around 1000 healthy people and fed them identical meals for two weeks while keeping track of their lifestyles and measuring their metabolic responses.

As well as the glycaemic response, it measured a class of fat called triglycerides, which can also spike in the bloodstream after eating. Again, the study found highly individual responses to identical meals. "Some people had hardly any rise, in others it dropped back fast, in others it was going up and up for hours," says Spector. But triglyceride spikes weren't correlated with glucose spikes. "Everyone reacts differently to identical foods," says Spector.

Triglycerides are a risk factor for chronic diseases too. "If you've got all these fats circulating in your blood for long periods of time, it increases inflammation and you get metabolic problems, diabetes, heart disease and obesity."

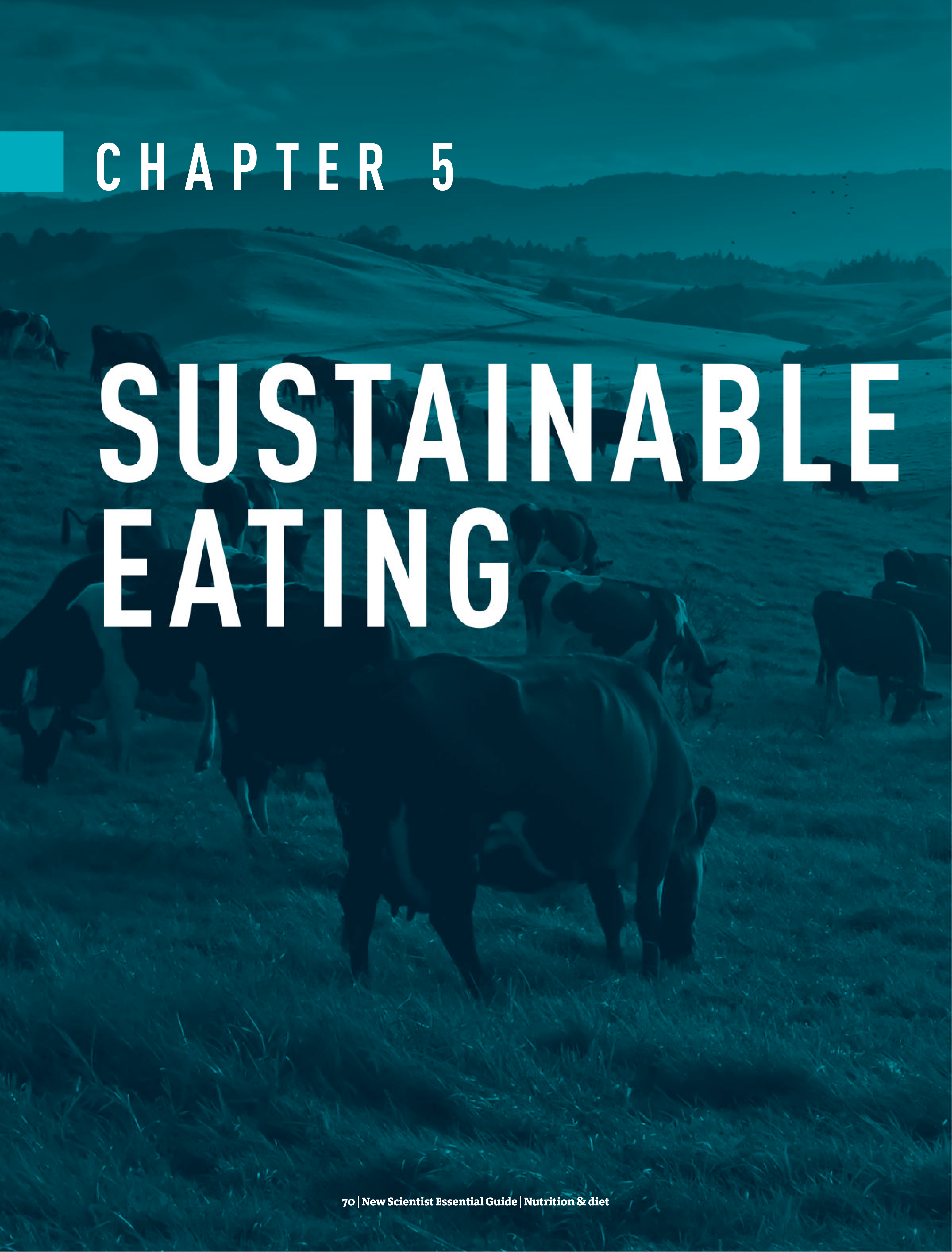
After crunching all the data, Spector's team found that an aggregate of several measurements, including age, sex, height, blood pressure and microbiome, could fairly accurately predict an individual's metabolic response to any given meal. It is far from perfect, but is still progress from merely recommending a universal healthy diet.

In the future, maybe you could visit your doctor, donate some blood, stool or urine, take a few tests and go home with a precision diet plan tailored to your individual needs. Until then, there are a few things you can do at home to work out which foods are good and bad for you.

About 10 per cent of people can feel themselves having sugar highs and lows, says Spector, so if certain foods give you a sugar rush then a crash, avoid them.

Elinav recommends using a skin-prick glucose sensor. "Try a food or meal and measure blood-sugar level afterwards: you'll get some idea what elements of your diet spike your blood sugar." That may be especially useful information for people who want to lose weight, says Sarah Berry, also at King's College London. "High peaks and big dips in blood sugar are related to hunger," she says.

"People who are bigger dippers get hungrier, consume more calories at their subsequent meal and consume more calories over the following 24 hours," says Berry. "Over a long period of time, that will impact weight. So we can look at personalising food to prevent that dip, which would then impact hunger, energy intake and weight." ▮



CHAPTER 5

SUSTAINABLE EATING

Whether you eat meat and fish or you are a staunch vegan, every food choice you make has an impact on the wider world.

The production of meat and dairy products in particular has a high environmental impact, in terms of its consumption of land, water, energy and other key resources. Eating less of both is a no-brainer if we want to eat more sustainably, and is probably healthier for us too.

Radically changing what we eat is easier said than done, however. How to persuade people to change their diet, and how to produce food more sustainably, remain contentious issues.

CHEESE'S DIRTY SECRET

Many people who turn vegetarian find themselves eating an awful lot of cheese. It's a truth that might be hard to swallow, but if you think that is better than meat for both animal welfare and the environment, you might need to think again.

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MANY of us have, at one time or another, at least thought about giving up meat and fish and going vegetarian. If you are one of those people, there is another villain on your plate that you may not have considered. Hard, soft, runny, blue, smelly – cheese.

The cheese industry is a gigantic success story. World production is at least 22 million tonnes a year, up from 15 million tonnes in 2000, and is projected to keep on expanding as people in traditionally cheeseless cultures in parts of Asia catch on. Of course, you don't have to be a vegetarian to love the stuff. But there is some evidence that, in some Western countries, a meat-cheese switch is happening. In the UK at least, studies show that people who don't eat meat consume more cheese than average.

That is a potential unreported problem for the environment. Animal farming is a major source of greenhouse gas emissions. According to the UN's Food and Agriculture Organization (FAO), the livestock sector produces 14.5 per cent of all human-made greenhouse gas emissions – on a par with all trains, cars, ships and planes. Cattle alone contribute two-thirds of this. Add in sheep, goats and buffaloes – the source of almost all the cheese not made from cow's milk – and that goes up to 81 per cent.

In terms of carbon footprint, beef is about as bad as it gets. Figures collected by researchers at the University of Michigan show that producing a kilogram of beef emits an average of 26.5 kilograms of "carbon dioxide ›

equivalent". This means it causes the same amount of warming over 100 years as 26.5 kilograms of pure carbon dioxide.

Lamb isn't far behind. Other meats, such as pork and chicken, result in lower carbon emissions, as do fish. But all of these are still carbon intensive compared with vegetables.

Milk is quite low, at 1.3 kilograms of CO_2 equivalent to produce a kilogram of the stuff. But cheese is many times higher, mainly because 10 litres of milk go into making each kilogram of cheese. The actual footprint varies a lot depending on the variety of cheese, but the mean is 9.8 kilograms of CO_2. The worst offender is cheddar produced in the US using milk from Holstein-Friesian cows, which comes in at more than 16 kilograms of CO_2.

On this basis, a quick calculation suggests that substituting cheese for some meats – say, halloumi for chicken – can double a meal's carbon footprint. But this is only a rough estimate, given that people might not eat the same amount of cheese as they would meat, and there are other environmental impact categories to consider than just carbon footprint.

Another way to look at the issue is the carbon footprint per calorie. By this measure, dairy products as a whole have a lower carbon footprint than any meat, so a straight calorie-for-calorie swap may mean that cheese is no worse than chicken. Unfortunately, the studies published so far don't look at cheese on its own, but bundle it into a general dairy category along with milk, cream, butter and yoghurt. And we don't know whether people really swap calorie for calorie as they tuck into the cheese board (gram for gram, cheese tends to contain more calories than meat), so it is impossible to be sure.

More clearly on the plus side, each calorie of dairy gobbles up less land and less energy than a calorie of meat. That is potentially good news for those who substitute cheese for meat – although, again, it isn't considered separately from other dairy products.

But even if we set aside the question of swapping meat for cheese, the environmental impact of dairy farming is a growing issue. "We are facing a lot of criticism," says Juha Nousiainen, a senior vice president of Valio, Finland's largest dairy company.

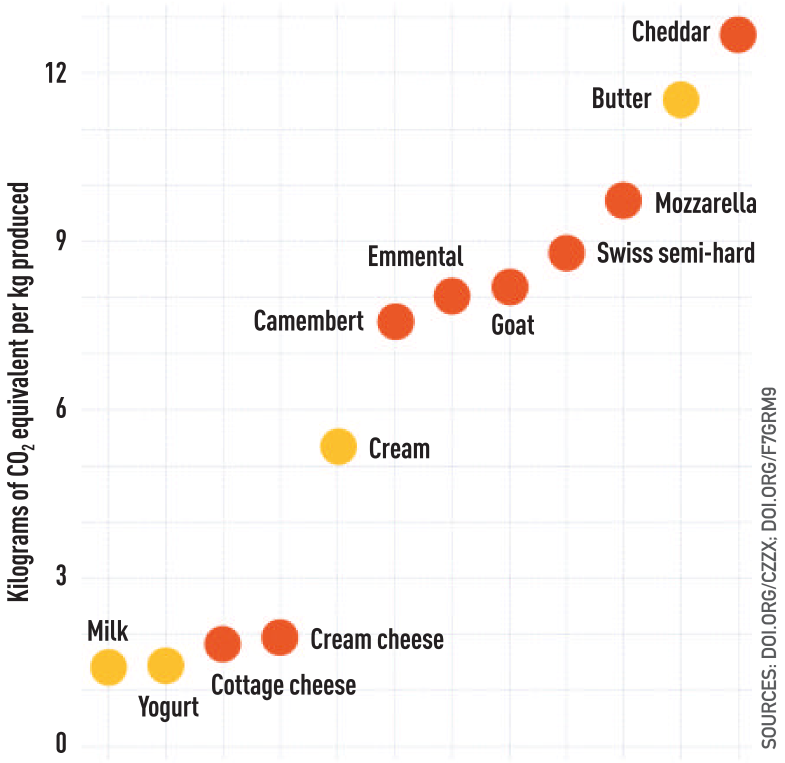
Valio's solution is the "zero-carbon cow" project. This is an extremely challenging goal, as methane production is inevitable and it can't be captured from cows in fields. The answer is to offset it: manage the pasture better so the grass becomes a carbon sink rather than a carbon source. That isn't as piddling as it may sound. According to the FAO, improved management of grazing land can sequester up to 3 tonnes of carbon per hectare per year – more than an equivalent area of boreal forest.

Even that, however, will only solve about half the problem. The rest will have to come from many small interventions, such as using manure as a biofuel and capturing the methane emitted inside barns. As yet, these are just plans on the drawing board. But if they work, "we can get very near to zero", says Nousiainen.

The environmental impacts are one thing. What about animal rights? For researchers, getting access to dairy farms to document how they operate isn't easy. In 2010, Kathryn Gillespie, a geographer now at the University of Washington, embarked on an investigation of the US dairy industry. It took her four years to gather enough material to publish, but what she found is shocking. Her peer-reviewed book *The Cow With Ear Tag #1389* – named after a wretched animal she encountered at a cattle auction – describes the highly industrialised process of milk production and the toll it exacts on cows.

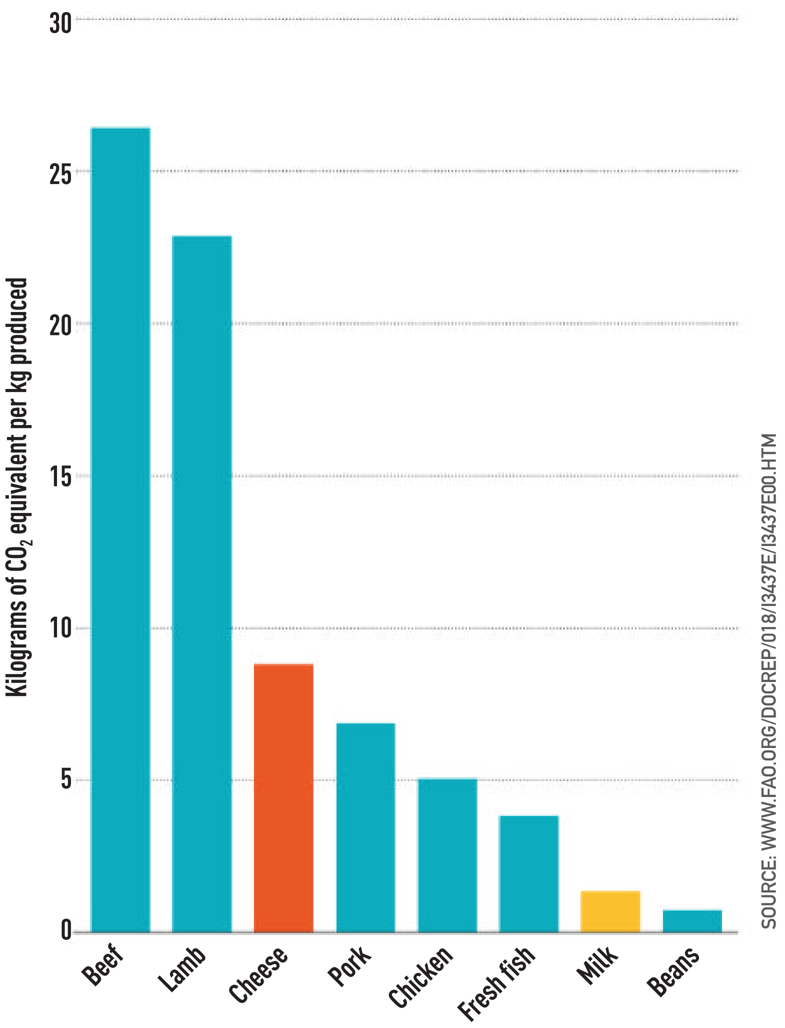
Most dairy cows are born on large dairy farms to adult dairy cows. They are separated from their mothers straight after birth, raised until they are about 18 months old, then artificially inseminated. In the meantime, they are dehorned, ear-tagged and, in some countries, have their tails docked.

Although not all cheeses have had their carbon footprints worked out, we know some are worse than others. So if you can't resist, pick yours wisely to avoid the worst offenders



SOURCES: DOI.ORG/CZZX; DOI.ORG/F7GRM9

Cheese has a higher carbon footprint than some kinds of meat



SOURCE: WWW.FAO.ORG/DOCREP/018/I3437E/I3437E00.HTM

Nine months later, they give birth. Their calves are taken off them immediately and they are then milked several times a day. To continue producing milk, they must be inseminated once a year, meaning they are pregnant most of the time. Female calves go into the dairy herd. Male calves are usually killed right away or reared for meat and slaughtered within six months.

Many of the welfare issues are exacerbated by intensification. Gillespie reports that, in the US, milk production per cow increased by 13 per cent between 2007 and 2016. This can cause an increased risk of the udder infection mastitis, hunger and lameness from carrying large amounts of milk.

The dairy industry is well aware of these issues. The European Dairy Association says it fully endorses the internationally recognised, science-based "five freedoms" set out by the World Organisation for Animal Health. These are: freedom from hunger, malnutrition and thirst; freedom from fear and distress; freedom from physical and thermal discomfort; freedom from pain, injury and disease; and freedom to express normal patterns of behaviour. However, there is ample research showing that these freedoms are routinely violated.

So what to do? Switching to sheep's or goat's cheese isn't the answer. They also belch out lots of methane. And, due to less intensive management, the carbon footprint of their milk is higher than that from cows: about 5 kilograms of CO_2-equivalent per kilogram. Goat farming also has welfare issues.

There are now plenty of plant-based cheese substitutes, but the taste and texture mean these vegan varieties are unconvincing and can bring environmental concerns of their own, such as the common use of palm oil, associated with deforestation. Ultimately, if you are concerned about the environmental and animal-welfare impacts of dairy, your best bet for now is to cut down or renounce cheese entirely. ▮

SHOULD WE ALL GO VEGAN?

If meat and dairy are both bad for the environment, that suggests we should all be moving towards entirely plant-based diets. People cite a range of reasons for going vegan, from improving health, to an opposition to animal cruelty and a desire to help cut greenhouse gas emissions. The question is, does it work for our health, too?

THE basic principle of veganism is to abandon all animal-derived products, particularly in food. That means no meat, fish, milk, cheese or eggs – even those laid by free-range chickens, which are still debeaked and can be packed as densely as nine birds per square metre.

There is little doubt that it is a good move for the planet. As we saw in the previous section, livestock farming is responsible for 14.5 per cent of global greenhouse gas emissions. According to the UN Food and Agriculture Organization, a quarter of the ice-free land on the planet is used to graze livestock. On top of that, a third of all cropland is used to produce additional food for them. Studies show that if we all went vegan, two of the biggest environmental problems – greenhouse gas emissions and clearing land for agriculture – would be slashed.

Some people argue that switching to vegan options – swapping your dairy latte for a soy version, for instance – still causes a lot of problems for the planet. It is true that soy plantations are a driver of deforestation, but tofu and soy milk aren't their main product. Animal feed is. So, even if we all replaced meat with soy, we would still see a huge decrease in soy production because so much of it is for animal consumption.

Good for the planet, then – but is it good for us? From a nutritional standpoint, if you are considering going vegan, you will need discipline to cover all the bases. Macronutrients are easiest. Vegan fats come from things like olive oil, nuts and avocados. Carbohydrates are in high supply in fruits, grains and beans. Protein is abundant in lentils, quinoa and tofu.

That said, plant proteins often lack essential amino acids present in animal-based proteins, such as those ›

HEALTHY YOU

It is possible to get what you need from plants alone, if you're careful

Omega-3s

Daily recommendation: 1.1 grams (women)/1.6g (men)

1/3 salmon fillet (40g) or **18** walnuts

Vitamin B_{12}

Daily recommendation: 2.4 micrograms

1/2 steak (110g) or **5.5g** enriched nutritional yeast

Calcium

Daily recommendation: 700 milligrams

100g cheddar cheese or **500g** cooked kale

SOURCES: NUTRITICS / NHS / NIH / USDA

HEALTHY PLANET

If you did go vegan, your ecological footprint would shrink

Protein

You need 45 to 55 grams of protein a day, which you can get from...

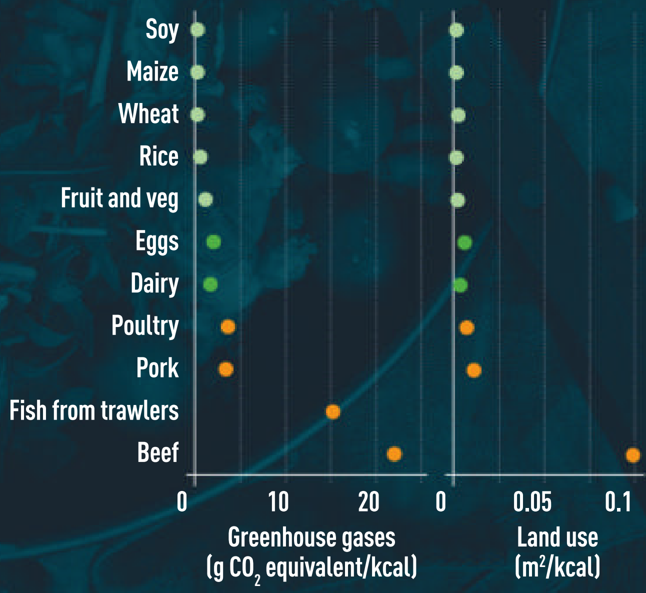
GREENHOUSE GASES *OR* LAND USE

1122g ⟨ 1 large pork chop (175g) ⟩ **3.5m²**

30g ⟨ 1 cup of cooked tofu (250g) ⟩ **0.6m²**

Greener veg

Vegan calories have a much smaller environmental impact than those from meat, fish, dairy and eggs



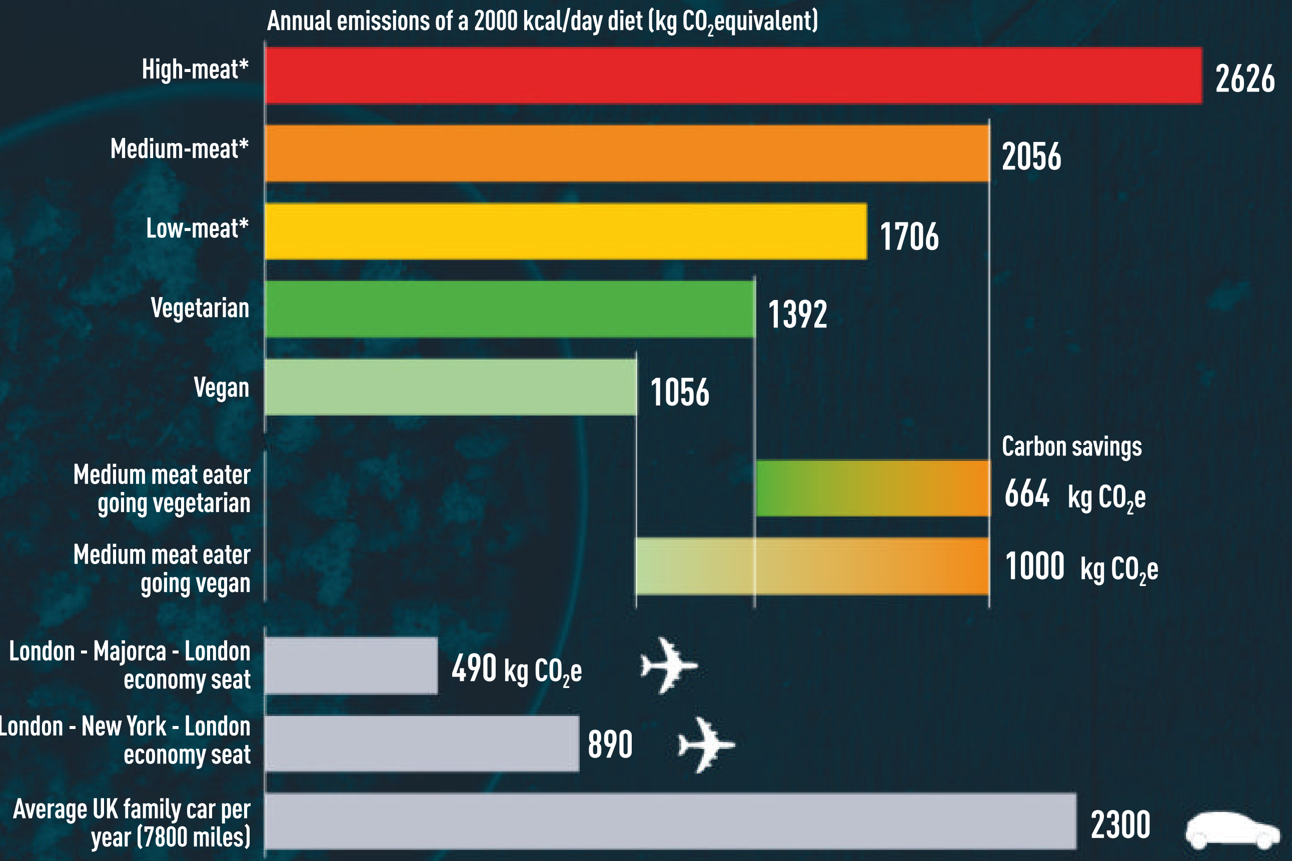
SOURCE: DOI.ORG/CJCW

Your choice

Most adults in the UK eat 110 grams of meat a day, making them high meat eaters. The carbon footprint of their diet is more than twice that of a vegan

A medium meat eater who decides to go vegan would cut their diet's carbon footprint by an extra 50 per cent compared with going vegetarian

Alternatively, you could just choose not to take that holiday in Majorca this summer



*High meat ≥100g/day *Medium meat = 50-99g/day *Low meat ≤ 50g/day

SOURCE: DOI: 10.1007/S10584-014-1169-1

found in milk, so vegans need to seek out alternative sources. Lysine, for instance, is an important building block of muscles and skin. It is plentiful in beans and legumes, and other essential amino acids can be found in seeds and chickpeas.

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Turn back to chapter 1 for more on the basics of nutrition

Like vegetarians, vegans don't eat seafood, so miss out on a convenient source of omega-3 fatty acids, which help build our cell membranes. That is why vegans have a healthy obsession with seeds and nuts. Chia and flax seeds, walnuts and leafy vegetables contain alpha-linolenic acid, an omega-3. Eating enough to meet daily recommended doses can be tricky, though, so an easier solution is to take supplements made from algae oils.

Supplements can also be useful for vitamin B_{12}. It is made by bacteria in the guts of some animals, like cows, and omnivores get it from eating meat. Like omega-3s, it is essential for our brains to function, and not getting enough of it can cause dementia, among many other problems. It is one of the most commonly cited nutritional challenges for vegetarians too, although one review found that B_{12} deficiency is more frequent among vegans. One way to fill the gap is with fortified breakfast cereals. Nutritional yeast is another good source. It may sound unappetising, but actually has a pleasant, slightly cheddar-like flavour.

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Chapter 3 has more on vitamins, minerals and omega-3

Without dairy, vegans must rely on green vegetables like broccoli and kale for calcium. Iodine, which is important for thyroid function and metabolism, can come from seaweed or cranberries. Iron is available in green vegetables, legumes, nuts and seeds. They contain a form that is harder for our bodies to use, so the US National Institutes of Health recommend that people who don't eat meat get nearly twice as much plant-based iron as those who do.

As for the overall effect on your health, evidence shows that, with some careful consideration, a plant-based diet can help you lose weight, lower your cholesterol, reduce your risk for certain cancers and even add a few years to your life expectancy.

This means a shift towards plant-based diets could bring economic benefits too. Marco Springmann at the University of Oxford estimates that if the entire world went vegetarian, we could avoid 7.3 million early deaths each year – 8.1 million if everyone were vegan. The healthcare savings would be worth more than $1 billion each year.

→
See pages 92 and 95 for more on plant burgers and artificial meat

If you can't contemplate the thought of never having another bacon sandwich or a creamy omelette, then you may be interested in the growing trend towards part-time veganism. Take Veganuary, where people give up animal products for January, for instance. The vegan before 6pm (VB6) diet is also gaining popularity.

A small pilot trial run by New Scientist and researchers at the University of Oxford, in which volunteers ate vegan food for a week and compared the benefits to a regular non-vegan week, found that the greenhouse gas emissions caused by their usual diet fell by 53 per cent during their vegan week. The participants consumed fewer calories, less saturated fat, carbohydrates and cholesterol, and their diets saw a significant increase in fibre. This suggests that regular forays into veganism, done carefully, could put you on the right track for long-term benefits, for your health and for the planet. ▮

GOING ORGANIC

While moving towards a more plant-based diet is undoubtedly good for us, the evidence of health and environmental benefits from the recent trend towards organic food grown without artificial fertiliser and pesticide is rather less clear-cut.

On the health side, it is as simple as saying that there is no convincing evidence that organic food is better for you than conventionally farmed food. On the environmental side, one huge problem is that organic farming requires far more land than conventional farming to produce the same amount of food. According to a 2017 study, going all-organic would require up to a third more land to feed the world by 2050, as opposed to 6 per cent more if we feed people conventionally. If climate impacts are high, 55 per cent more land will be needed with conventional farming – or 81 per cent if we go all-organic.

Even if the extra land needed for organic farms could be balanced out by land freed up by cutting meat consumption, this might not be our first priority for expansion. Land is ever more precious and as our farms, cities and roads grow, we are seeing the beginning of a mass extinction driven partly by habitat loss. In addition, limiting warming to under 3°C now requires removing vast amounts of carbon dioxide from the atmosphere. The best way to do this is to capture the carbon via reforestation and biomass burning – and this will require vast amounts of land.

A large meta-analysis on the impacts of farming, published in 2017, broadly agrees with earlier ones that find organic is no better in terms of greenhouse gas emissions. It specifically concludes that the best way to reduce the environmental impact of farming, besides eating less meat, is to improve conventional methods rather than switch to organic farming. One way to do that might be to embrace genetic modification methods to increase crops' pest or climate resistance. Organic farming explicitly rejects such methods, which is another reason to be cautious about advocating it as a large-scale solution.

THE RISE OF ALT-MILK

Milks made from peas, nuts and more are taking supermarket shelves by storm. But are they as good for you and the planet as you think?

BETWEEN 2012 and 2018, non-dairy milk sales in the US rose by 61 per cent, according to market research by Mintel – and that trend has only accelerated since. More than half of the market is taken up by almond milk, with soy and coconut milks making up another quarter. Oat milk is also growing in popularity. Manufacturers position the products as a healthy alternative, both for the body and the planet. But is that really true?

Nutritionally, it depends on which milk replacement you consider. In general, they are made by grinding up plants and soaking them in water, then adding emulsifiers and stabilisers to thicken the liquid and keep it from separating, but they have a lot of variety.

In terms of protein, soy milk is quite like cow's milk and it contains similar omega-3 fatty acids that are important for heart health. Almond and cashew milks have less than half the calories found in cow's milk, but are lower in protein. Coconut and hemp milk have a rich texture owing to their high fat content and they also include a small amount of dietary fibre not found in cow's milk. Oat and rice milks are higher in

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carbohydrates than both cow's milk and other plant-based alternatives.

Milks made from legumes, such as peas, soya beans and peanuts, also offer amino acids not found in cereal crops. Each type of alternative milk has its nutritional benefits and limitations. Any one of them can be considered healthy only when combined with a well-rounded diet, though the same can be said for dairy milk.

But what about their carbon footprint? In this respect, rice milk is one of the most environmentally costly alt-milks to produce. When rice paddies are flooded to stimulate plant growth, submerged biomass decomposes without oxygen, producing the potent greenhouse gas methane.

Of course, cows are notoriously bad for the environment as well. The carbon footprint of producing cow's milk varies from place to place, but in Western countries, it is typically around twice as big as that of making plant-based alternatives. A 2010 report by the Food and Agriculture Organization of the United Nations found that the production, packaging and transportation of cow's milk emits 4 per cent of all human-caused greenhouse gas emissions.

But carbon isn't the only environmental concern to consider. Nuts are notorious water sinks, with some requiring nearly as much water to produce as cow's milk. In a 2010 report, the UNESCO Institute for Water Education assessed the water footprint of global food crops. It found that of the plants used for alt-milks, water usage was highest for nuts, flax and soy, and lowest for coconut, oats, rice and hemp.

On the whole, it is clear that alt-milks are friendlier to the environment than traditional dairy, but their growing popularity may cause problems. If demand for coconut milk skyrockets, for example, it will become more profitable to grow coconut trees, which could lead to deforestation as farms expand. To avoid that, it is best to have several non-dairy options to choose from. So maybe the proliferation of alt-milks is a saving grace after all. ∎

FANGXIANUO/ISTOCK

Alternative milks promise a lower carbon footprint than conventional dairy milk

NO SUCH THING AS A (SUSTAINABLE) FISH

Labels declaring seafood "sustainable" give us comfort we're consuming ethically, but tracing the true environmental toll tells a different story. When it all is said and done, it's hard to find any fish that we can catch without having some negative impact on the environment.

MUCH of the seafood you see on sale at the supermarket will bear a label certifying that it was caught or farmed sustainably, or at least "responsibly". But what does that mean? Can you eat fish with a clear conscience?

Every year, we collectively eat more than 155 million tonnes of fish, about half of it wild-caught and half farmed. And consumption is growing. Since 1950, the human population has grown by about 175 per cent. In that same time, the amount of fish we eat has increased by 750 per cent.

It seems a fair bet that the impact of that trend on fish stocks isn't good – but it is surprisingly difficult to arrive at clear numbers. "Many countries do not have research ships to go to sea and monitor the stocks," says Manuel Barange at the United Nations' Food and Agriculture Organization (FAO). Even when they do, the science is challenging. It requires an estimate of the total biomass of a species within a huge geographical area, and then an assessment of whether that is enough to support the maximum sustainable yield. The margin of error is so large that a stock is considered sustainable even if it is 20 per cent lower than needed for the maximum sustainable yield.

Even with this wiggle room, the FAO says that about a third of the fish stocks it monitors are overfished, and hence on the road to collapse. In 1974, when the FAO first started counting, 90 per cent of stocks were sustainable. Today, just 65 per cent are.

Another major concern is that sustainable doesn't necessarily mean environmentally benign. Large-scale commercial fishing, which began in earnest in around 1950, can have many negative impacts on the wider ecosystem, such as the accidental catch of non-target species, called by-catch. A recent study by WWF concludes that it kills more than a million marine mammals, reptiles and birds every year. Lost or

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PAOLO CIPRIANI/ISTOCK

The amount of fish consumed globally has increased by 750 per cent since 1950 discarded fishing equipment is also a problem, killing untold numbers of marine animals that get caught up in it.

Certain fishing methods can also take a toll. Bottom trawling, where nets are dragged along the seabed, indiscriminately disrupts and damages marine habitats, possibly even contributing to pollution by undermining the ability of sea-floor microbes to remove harmful sediments.

Even the concept of sustainability has been questioned. You can actually overfish sustainably. It is like cutting an immense forest, but leaving a few trees standing, which you then harvest sustainably.

Looking instead at the current biomass compared with that in the water in 1950 shows that nearly all the world's fish stocks are profoundly depleted. The biomass of big fish has diminished to the order of 80 to 90 per cent.

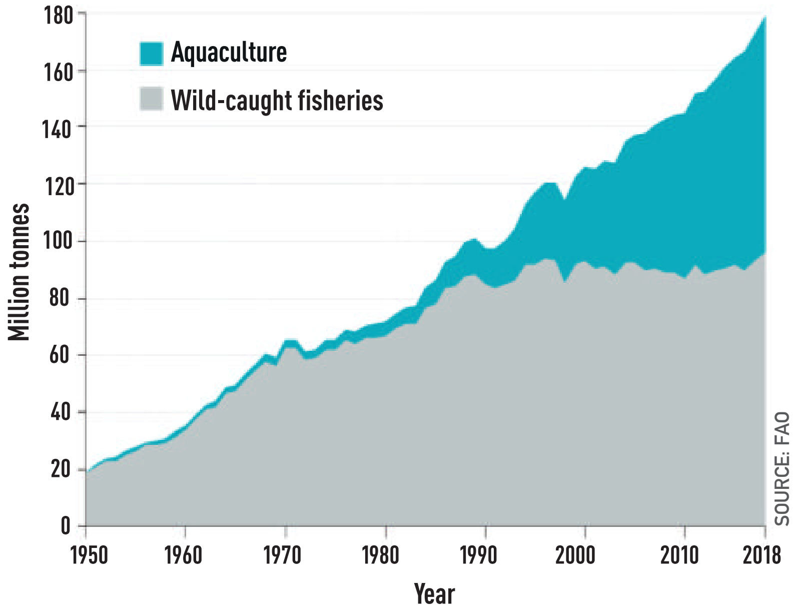
Sustainability also often fails to take into account wider ecological factors. The langoustine fishery in the Firth of Forth in Scotland, for example, is sustainable, but only because so many other species have been fished to extinction and the langoustines no longer have any natural predators. There is also the greenhouse gas emissions of wild-fishing operations to consider. According to a recent assessment, per kilocalorie of food produced, wild-caught fish has a bigger global warming footprint than pork, chicken or dairy.

Maybe, then, the answer is aquaculture, aka fish farming. This large and rapidly growing sector already supplies 52 per cent of the fish consumed directly by humans, and is projected to increase as demand for seafood rises but the catch from the wild stays essentially flat.

Aquaculture is the fastest-growing sector of global food production. The vast majority happens in Asia, largely for local consumption. Western consumers mostly encounter it in the form of farmed salmon or shrimp. For those consumers striving to make ethical choices, that can spell trouble.

Fish farming has some well-known and undeniable problems, says Grant Stentiford at the Centre for Environment, Fisheries and Aquaculture Science in

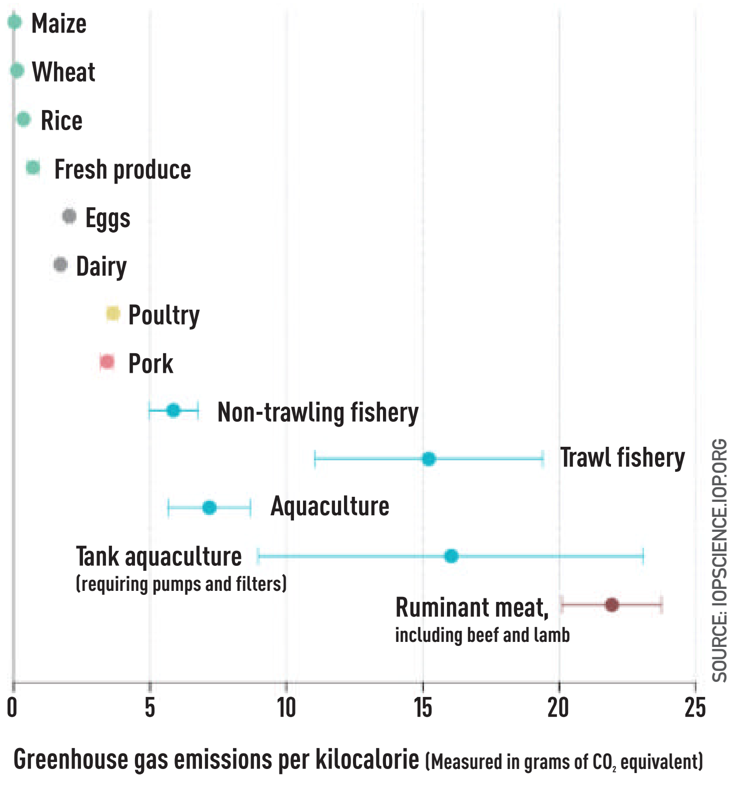
Aquaculture is rapidly expanding to satisfy the world's appetite for seafood



SOURCE: FAO

NOTE: Excludes aquatic mammals, crocodiles, alligators and caimans, seaweeds and other aquatic plants

Only ruminant meat such as beef generates more greenhouse gas emissions than farmed and wild-caught fish



SOURCE: IOPSCIENCE.IOP.ORG

Weymouth, UK. Farmed shrimp, for example, mostly comes from southern and South-East Asia and Ecuador, reared in ponds that were created by destroying mangrove swamps. "There has been a loss of habitat and biodiversity in relation to those industries. I don't think anyone can really argue about that," says Stentiford. Add in the environmental cost of feeding the shrimp and freighting them to Western markets, and their calorie-for-calorie carbon footprint can sometimes exceed that of beef.

Salmon farming, meanwhile, has well-publicised problems with parasites, the overuse of antibiotics, escaped fish breeding with wild ones – potentially diluting the gene pool of wild fish and, in some cases, leading to sterile offspring – and the pollution of the sea floor underneath the pens.

Another huge problem with aquaculture is that, paradoxically, it often increases the pressure on wild fisheries. Salmon, tuna, sea bass and many other farmed species are top predators that eat other fish. To meet this demand, around 22 million tonnes of wild fish are caught each year and processed into fish meal. Most of these are sardines, anchovies and other small fry that are edible for humans. To make matters worse, they are mostly caught in the waters of low-income countries, which often have food security issues, and then exported to richer countries.

With all these layers of complexity, how do consumers make informed choices? There are several accreditation schemes for wild and farmed fish, but they are far from comprehensive. One of the best known is the Marine Stewardship Council (MSC), which prides itself on its stringent sustainability standards and tracking of supply chains.

However, just 16 per cent of the world's wild-caught fish is landed by MSC-certified fleets. The rest may or may not be sustainable, or may not have been assessed by an oversight body. It is impossible to know. What's more, the MSC currently takes no account of greenhouse gas emissions or animal welfare. Even fisheries scientists can struggle to know what to buy. The only thing we can be certain of is that if you do eat fish, there is almost certainly something fishy about it. ▮

FOOD WASTE

Whatever diet we adopt for ourselves or for the planet, here is a startling fact to bear in mind. In 2019, around 931 million tonnes of food went into the waste bins of households, retailers, restaurants and other food services – something like a sixth of all the food we produce.

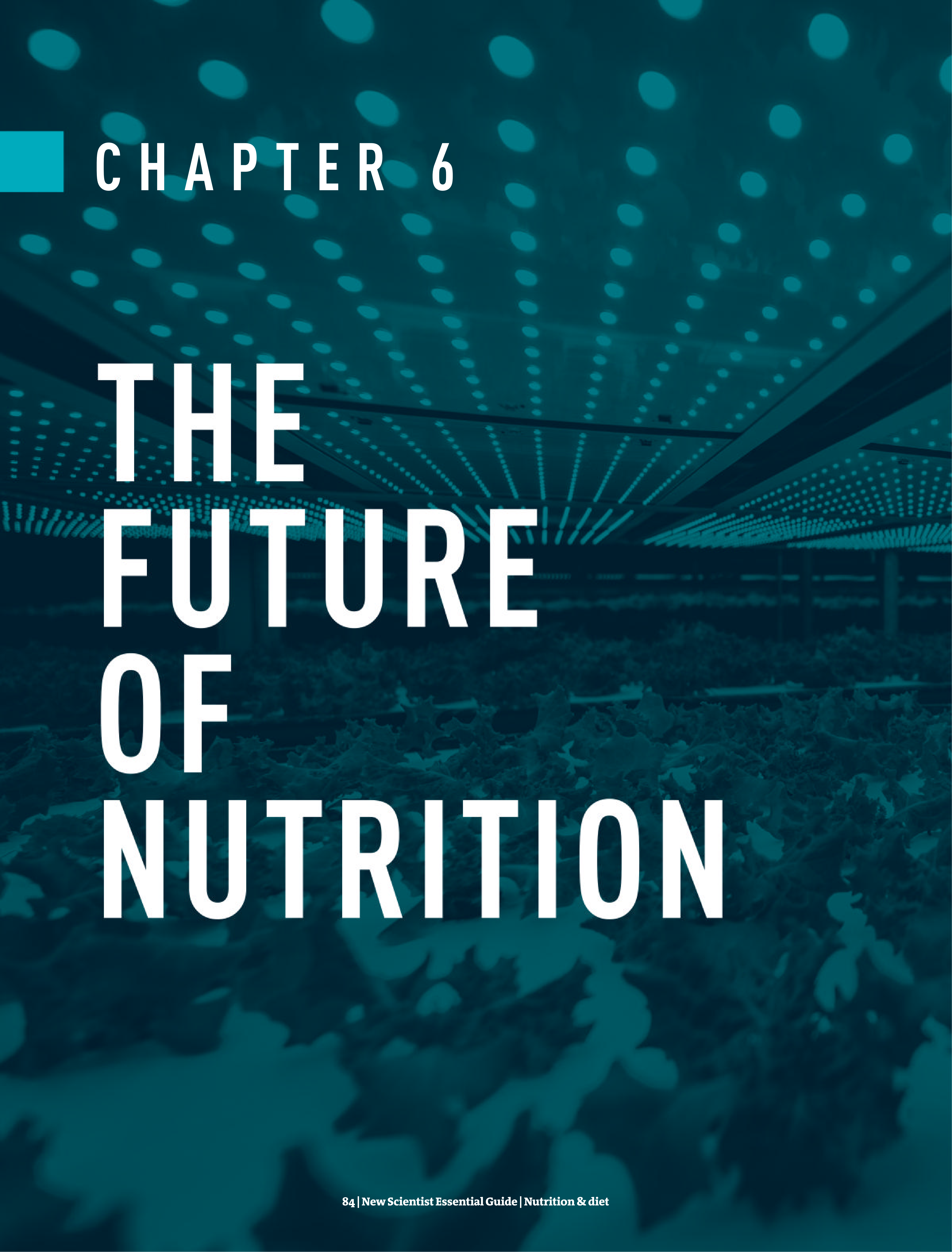
That is the conclusion of the 2021 Food Waste Index Report produced by the United Nations Environment Programme and UK charity WRAP, which promotes sustainability. The study looked at food waste in retail outlets, restaurants and homes by reviewing government data and academic studies across 54 countries with a mix of incomes. Most of this waste – 11 per cent of all the food produced – occurs in homes. Globally, the average person throws away 121 kilograms of food, with 74 kilograms of this happening within households.

Food waste isn't just a problem in high-income countries, the report found. On average, annual per capita food waste in homes is 79 kilograms in high-income countries versus 91 kilograms in lower-middle-income countries.

The report included both edible and non-edible waste, such as bones or vegetable peelings that must be thrown away. Lower-middle-income countries may have higher per capita food waste because food is more often prepared from scratch, which might increase inedible food waste.

Food losses pre and post-harvest and food waste by consumers have a substantial impact on the environment and climate change, making up around 9 per cent of global greenhouse gas emissions. It also increases demand on land for agriculture and raises water use.

Reducing food waste is vital and all individuals have a part to play, says Richard Swannell at WRAP. "Rather than focusing on supply chains, we need to tackle food waste inside homes with behaviour change campaigns."



CHAPTER 6

THE FUTURE OF NUTRITION

The food on our plates in a few decades' time is likely to look very different to today. The sustainability of what we eat is only going to become a more pressing question, creating new imperatives to find alternatives to meat and to develop new crops that are less demanding of land, water, energy and other resources.

Feeding into that is our evolving knowledge of what we actually need to eat for longer, healthier lives. Even though we have made great strides in our understanding of this, there is still much that we don't fully grasp – and the question of whether we will find new foods produced via technology palatable remains.

NUTRITION'S DARK MATTER

It might be an unpalatable way to think about it, but ultimately food is just a mass of different chemical compounds. Astoundingly, we don't know what 99 per cent of them are, how they interact or what their effects might be on our health. Now researchers are setting out to change that.

ELIJAH_SAD/ISTOCK
PREVIOUS PAGE: AZMANL/ISTOCK

"OUR understanding of how diet affects health is limited to 150 key nutritional components," says Albert-László Barabási at Harvard Medical School. "But these represent only a small fraction of the biochemicals present in our food."

It was Barabási who coined the term "nutritional dark matter" for all the rest, in analogy to the 85 per cent or so of material in the universe that physicists say is there but can't be observed directly. There are thousands and thousands of compounds that are in food but were, until recently, totally unknown, and that may be affecting our health

The official source of information on this complex biochemical soup is the National Nutrient Database for Standard Reference, maintained by the US Department of Agriculture (USDA). It contains information on the composition of hundreds of thousands of foods, broken down into 188 different nutritional components.

Search for "garlic", for example, and the database serves up 58,055 foodstuffs that contain it, ranging from whole, raw garlic to processed foods such as instant noodle soup. The entry for raw garlic lists 67 macro- and micronutrients, some quantified down to micrograms per 100 grams or concentrations of less than 0.00001 per cent. That may seem detailed, but even this is far from comprehensive. The USDA database tracks only common nutritional components in many foods, and so omits many rarer ones. For example, it leaves out some of the quintessential flavour compounds in garlic, such as alliin.

Around 10 years ago, an international team of researchers decided to compile a more comprehensive database after trying and failing to get detailed information on various foods' composition. David Wishart at the University of Alberta, Canada, and his colleagues trawled the literature and filled in other blanks through their own chemical analyses in the lab.

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The result is a massive database called FooDB, which Wishart says now holds information on about 70,000 nutritional compounds, nearly 400 times more than the USDA database. For raw garlic, that amounts to 2306 compounds.

Why go hunting for the components of our food in such detail? Principally, because there is good reason to believe that some of the neglected compounds have meaningful effects, both for good and ill, says Larry Parnell at the Jean Mayer USDA Human Nutrition Research Center on Aging at Tufts University in Massachusetts. This idea hasn't gone down well in some established nutrition circles, with some believing that the nutrients we are fully aware of account for almost all the impact that diet has on health.

An obvious objection to the claim that understanding this nutritional dark matter is important for health is that most of the compounds occur in tiny quantities. But concentration isn't always a factor: vitamin E comes at micrograms per 100-gram intake of food, yet its absence has adverse health effects.

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See chapter 3 for more on vitamins and minerals

For another counter-argument, let's talk about steak and garlic. Gut bacteria break down a substance in red meat to produce a compound called trimethylamine (TMA). This is then absorbed and transported to the liver, which converts it into trimethylamine N-oxide (TMAO). High levels of TMAO in the bloodstream are linked to significantly higher death rates from coronary heart disease. But one of garlic's flavour molecules, allicin, is known to inhibit TMA production. This may be a reason why the garlic-rich Mediterranean diet seems to protect against heart attacks, rather than other factors, such as it being low in saturated fats. Allicin has also been reported to have anticancer properties, while luteolin, another component of garlic that isn't in the standard database of food compounds produced by the USDA, also seems to protect against cardiovascular disease.

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Turn back to page 24 for more on the Mediterranean diet

To start to understand the scale of our ignorance, Barabási and his colleagues consulted another source – the Comparative Toxicogenomics Database, an inventory of how thousands of chemicals interact with our bodies. First, they looked at the 67 "official" components of garlic and found that 37 were already known to be linked to human health and disease. Then they did the same for the 2306 compounds in garlic listed in FooDB and discovered a further 574 with potential health effects.

These vast tracts of uncharted complexity could be the reason why nutrition science so often produces inconsistent and irreproducible results. Without a complete picture of the nutritional composition of food, you can't be sure you are comparing the right things.

This is also true of individual micronutrients. "Consider beta carotene," says Barabási. "It tends to be positively associated with heart disease, according to epidemiological studies, but studies adding beta carotene to the diet do not show health benefits. One potential reason is that beta carotene never comes alone in plants; about 400 molecules are always present with it. So epidemiology may be detecting the health implications of some other molecule."

Added to the complexity is the fact that most dark nutrients are chemically transformed by your gut bacteria. Not controlling for variation in gut microflora means people get different doses of metabolites from their food.

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Turn back to page 16 for more on the gut microbiome

As if all that wasn't hard enough, there is the added complication that cooking transforms chemical components into other ones, sometimes with health implications. When sugars and amino acids react at high temperatures, the result is new molecules that make roasted and grilled food delicious, but the reactions also produce the compound acrylamide, which is a probable human carcinogen. Cooking and processing nutritional dark matter could produce similar but unknown toxins, says Barabási.

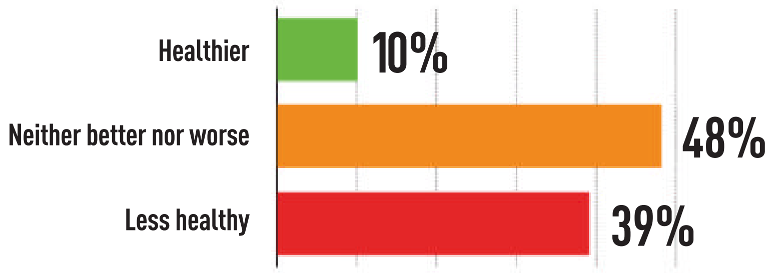
There are also the myriad chemicals added during food processing. What happens to them when they encounter our metabolisms and microbiomes is unknown. If just identifying the compounds in food is a challenge, working out how they interact with human biology is an even bigger one. "Ultimately, we need to connect all the food molecules to their molecular effects in the human cell," says Barabási.

All of which suggests there is a food mountain to climb to understand the interplay between nutrients and human health. It will require yet more data on individuals' genomes, microbiomes and other factors, but it is perhaps the only way for us to truly understand the complexities of diet. "So much to do," says Barabási. "But we're making progress." ▮

GENETICALLY MODIFIED FOOD

Genetically modified foods have been around for decades, but there has been no reason for consumers to be keen on them. Virtually every GM crop on the market is designed to help the farmer who grows it rather than the person who eats it. Now that's starting to change – with a new generation of GM foods designed with health or flavour in mind. The odd thing is, no one plans to tell you about them.

Few people in the US believe GM foods can be better for their health than non-GM foods



SOURCE: PEW RESEARCH CENTER (2016)

THE first ever genetically modified (GM) food to go on sale – the Flavr Savr tomato, launched in 1994 – was designed to stay fresh for longer. This meant it could be picked after ripening and thus tasted better than normal supermarket tomatoes, which are picked green and ripened artificially at the expense of flavour. But it was discontinued after three years because it wasn't profitable.

Other GM crops – ones that have had DNA from another organism introduced into their genome to encourage beneficial traits or suppress undesired ones – have thrived, however. Most of the soya grown globally, and more than a quarter of maize and oilseed rape, is now GM. In some countries, such as the US, most processed foods contain at least one ingredient, for example corn syrup, derived from GM crops. These typically have traits whose benefits, such as insect or herbicide resistance, are obvious only to farmers. But in the past few years, this has started to change, with foods arriving that offer added perks for consumers.

The Innate potato, for instance, is less prone to bruising and the resulting black spots, and produces less of the potentially dangerous chemical acrylamide when fried than conventional potatoes do. It has been sold as White Russet in the US since 2015. Then there are apples that will appeal to those people – especially children – who don't like eating fruit that has gone brown after being sliced. Arctic apples, which never discolour, went on sale in the US in November 2017. Other prospects include a coeliac-friendly wheat that contains only "good" gluten, higher-fibre white bread and more.

So far, these products are being produced in relatively small quantities. But the next GM food to make its way onto our plates – or into our takeaway boxes – is going to be huge.

Soybean oil is incredibly popular, especially in the catering industry. Yet it isn't actually ideal for cooking. The raw oil consists of around 60 per cent polyunsaturated fats, which are unstable. These limit the oil's shelf life and break down when used for frying to form bad-tasting and harmful by-products.

By contrast, monounsaturated fats like oleic acid, found in olive oil, are more stable and thus better for cooking. But olive oil, which is up to 80 per cent oleic ›

THE CRISPR REVOLUTION

CRISPR gene-editing techniques promise advances such as berries that are easier to pick

GWENVIDIG/ISTOCK

Critics of genetically modified (GM) crops say they were supposed to have all kinds of benefits, from boosting food production to helping the environment, but haven't delivered. That is probably wrong. For instance, a 2018 meta-analysis found that GM maize yields are up to 25 per cent higher. Several reports, including one in 2016 from the US National Academies of Sciences, also suggest that GM crops are no worse for the environment than conventional ones and are sometimes better.

What is true, however, is that most GM crops are designed to enrich big companies. That is because on average it takes 13 years and costs $130 million to launch one, meaning only big multinationals have traditionally had deep-enough pockets to develop them.

That's changing now with the advent of CRISPR, a gene-editing technique that has made genetic tinkering cheap and easy. Older genetic modification techniques add extra DNA to a plant or animal, often taken from a different organism. By contrast, the most common use for CRISPR is to change just one or two DNA letters in what is already there. This simpler approach has resulted in an explosion of companies all over the world coming up with improved crop varieties.

Genome-edited plants and animals could make what we eat safer by removing allergens and cancer-causing substances such as acrylamides. CRISPR could also make crops more nutritious and resistant to diseases. They could even make berries easier to pick by machinery, the cutting costs of manual labour.

That has become all the more vital, as we need the next generation of food to tackle challenges such as a growing population and climate change. By opening biotech up to small companies, CRISPR offers us a chance to avoid the mistakes made with genetically modified organisms (GMOs) and to ensure more people benefit. Whether that happens or not depends on if regulators see CRISPR as a smarter form of conventional breeding or treat it as a new form of GM.

All breeding involves genetic modification of some kind. Farmers who select a prize cow to mate are choosing to propagate its genes. Such mutations occur with no human intervention all the time, so CRISPR proponents argue that there is no logical reason to treat them differently. In fact, plant breeders in the 1950s gave up waiting for natural mutations and started inducing them with radiation or toxic chemicals, called mutagenesis. Much of our food was created in this way, such as some wheat varieties and red grapefruit.

Anti-GM activists disagree. They want all edited crops to be treated as GM. Only a few countries have made their position clear so far. In the US, crops created by minor gene-editing tweaks that could have been produced with conventional breeding are treated as normal, but animals created in the same way are subject to additional regulation. The EU, by contrast, currently rules that gene-edited crops should be regulated as GMOs. It has, however, recently reviewed its position, opening the door to a possible loosening of restrictions.

As for the rest of the world, Canada, Argentina, Brazil and Chile will regulate on a case-by-case basis, with most edited varieties being treated as normal ones. The UK, previously bound by EU decisions, is due to publish the results of its own consultation on gene-edited food in 2021.

acid, is expensive. Back in the 1950s, producers started taking cheap-as-chips soya oil and hydrogenating it, a chemical process that makes it more stable. By the mid-1960s, more than half the cooking oil sold in the US was hydrogenated soybean oil. Today, it is the second-most widely used vegetable oil in the world after palm oil.

Hydrogenation solved the cooking problem, but it has a serious health downside: it causes trans-fatty acids, or trans fats, to form. By the 1990s, it was becoming clear that these greatly increase the risk of heart attacks and strokes. In 2003, Denmark became the first country to ban them.

For decades, companies have been working to produce cheap, trans-fat-free oils by altering crops so that their oil contains more oleic acid. Two US firms have created genetically engineered strains of soya that are high in oleic acid: Monsanto, with one called Vistive Gold, and DuPont Pioneer with Plenish. Their oils are more than 70 per cent oleic acid – similar to the amount in olive oil.

Still, these oils remained a niche product until 2015, when the US Food and Drug Administration ruled that trans fats had to be phased out of all foods over the following three years. "We realised that this trans-fat ban was going to make big changes in the food industry," says Federico Tripodi, head of Minnesota-based company Calyxt, which has developed a high-oleic soybean using gene editing. "The soybean industry estimates the long-term market potential for high-oleic soybeans is 15 million to 20 million acres," says David Tegeder at DuPont Pioneer. That would make them one of most widely grown crops in the US – and would mean that many millions of people around the world will be consuming their oils.

On the face of it, that is good news. Besides having less of the trans fats, high-oleic oils are also much less likely to break down into aldehydes when used for cooking – and these are even more toxic than trans fats. A third plus is that the oils contain less saturated fat, so should reduce the risk of heart disease. You might expect, then, that the companies behind these products would be keen to extol their virtues to consumers, especially in light of the bad reputation that GM foods have in some quarters. Far from it – the firms involved seem to be taking a softly-softly approach. It is local labelling laws that will determine whether consumers can tell if they are buying or eating food containing these oils.

Restaurants – especially fast-food ones – are likely to embrace them. As well as complying with the trans-fats ban in the US, high-oleic soya oils have an extended shelf life. Yet restaurants have little to gain from advertising the switchover, given that many people remain opposed to GM. Both Plenish and Vistive Gold have been approved in the EU, where GM food has to be labelled on menus, but even there consumers are likely to remain none the wiser. Anti-GM campaigners say many restaurants already routinely use GM cooking oil without declaring it, perhaps because they may not be reading the small print when they buy the oil in bulk.

What about your shopping basket? GM foods don't have to be labelled in the US, so most people probably have no idea that the White Russet potatoes or Arctic apples they buy are genetically modified. In fact, a 2013 survey found that half of people in the US didn't even know if supermarkets sold any GM foods.

Things are changing. In 2018, a new US labelling law instructed firms to add a scannable barcode to any food containing GM ingredients. However, food made from gene-edited crops won't have to be labelled. The argument here is that varieties of plant created by altering their own genes, rather than by adding extra genes sometimes taken from other organisms, are distinct from normal GM crops. US regulators appear to agree. For instance, they have told Calyxt that its products, such as its high-fibre wheat and its good-gluten flour – which is made by slicing out the genes that make the kind of gluten implicated in coeliac disase – will be treated as conventional rather than GM foods. The EU is still vacillating. A recent report concludes that current GM legislation, adopted in 2001, isn't fit for purpose, and the bloc has begun an open consultation process to create a new legal framework for these technologies.

Yet even if GM foods that benefit the consumer are clearly labelled and marketed as being modified, there is no guarantee this will change attitudes. "I think it's going to be difficult to change people's minds," says writer and environmentalist Mark Lynas, an anti-GM campaigner turned supporter.

The stakes are high. GM crops could help us produce better foods in a more environmentally friendly way, which will be ever more important as the population grows and the planet warms. For instance, Napier is developing crops rich in beneficial omega-3 oils. As these oils typically come from wild fish caught to feed farmed fish, this could make fish farming more sustainable as well as having health benefits.

←
Turn back to page 81 to read more about the problems with fish

The arrival of a generation of GM crops with clear benefits for consumers should be big news. But far from helping to win over hearts and minds, it seems few people will even realise what they are eating. ▮

PLANT BURGERS

Like meat, but don't want a side order of animal cruelty and environmental destruction? That's the motivation behind new foods created using the latest in biochemistry and technology that aim to mimic the taste, texture and nutritional qualities of meat with just plant fibres. But do they pass the taste test?

IF YOU love eating a grilled burger perfected into juicy, salty, fatty heaven... then you're probably struggling right now, painfully aware that as a population, we need to reduce how much meat we consume.

Plant-based meat analogues aren't your standard meat substitutes, such as tofu, Quorn and soya mince; nor are they the much-hyped but still experimental meats grown in the lab from stem cells. They are plant products processed to look, smell, taste and feel like meat – and they are aimed squarely at diehard meat-eaters.

↓
The next section has more on lab-cultured meat

San-Francisco-based firm Impossible Foods, maker of the Impossible burger, is a big player in this new market. "We're not going to be able to just talk people into a plant-based diet," says its chief scientist David Lipman. "We need to create foods that are so delicious that people choose them instead of animal-based foods."

This is a big challenge. The first step is getting to the heart of what makes meat so delicious. When meat is cooked, it exudes a complex mixture of volatile compounds that give it an unmistakable aroma. This is what we mean when we talk about the flavour of a steak or burger, because so much of what we experience as flavour is actually aroma tickling the olfactory receptors lining the top of our nasal cavity. For Lipman, recreating this signature aroma of cooked meat is vital.

←
Turn back to page 17 for more on what taste is all about

Impossible is going through the painstaking process of trying to identify all of those compounds, deciding which are the most important and then finding plant analogues.

Lipman's team realised early on that the key ingredient in meat's flavour is haem, an iron-containing compound found in blood and muscle tissue that gives meat its colour and metallic tang.

Haem isn't only found in animals. Some plants contain an identical molecule bundled up in a different protein, leghaemoglobin, which has the same rich flavour and colour as animal haem. One source is the roots of legumes like soy, but pulling up plants to harvest it would be a costly and difficult process. The solution was to genetically engineer yeast to produce leghaemoglobin. ›

GOOD FOR THE PLANET

Plant-based meat substitutes have a much smaller footprint than the real thing

	IMPOSSIBLE BURGER (85 grams/3 ounces)	20% FAT BEEF BURGER (85 grams/3 ounces)
CARBON FOOTPRINT (kg CO_2 equivalent)	0.6	2.4
WATER CONSUMPTION (Litres)	17	68
LAND USE (square metres)	0.31	6.2

LESS SO FOR YOU

Cholesterol apart, fake meat is little better than beef

CALORIES	220	213
TOTAL FAT (daily requirement)	13g (17%)	16.8g (26%)
SATURATED FAT	10g (50%)	6.4g (32%)
CHOLESTEROL	0	60mg (20%)
SALT	1.08g (19%)	0.14g (2%)

SOURCES: IMPOSSIBLE FOODS; USDA

With haem cracked, Impossible moved on to other elements of the meat experience. Its burger gets its sizzle and char from small flecks of coconut oil scattered throughout the mix. This fat is solid at room temperature, melts in the right way and has a neutral flavour. Wheat and potato protein add texture, while yeast extract and soy protein impart more umami flavours. The whole edifice is held together with plant gums, with spices and seasoning adding the finishing touches.

Another US brand, Beyond Meat, has a similar burger for sale in shops such as Whole Foods. The technology to create its texture involves state-of-the-art protein extrusion, which forces mixtures of vegetable proteins through a narrow aperture to create meat-like fibres.

Other brands are trying different approaches. Moving Mountains, based in the UK, launched its B12 burger in 2018. Its main ingredient is mushroom, alongside wheat and pea proteins. "We first analysed how beef was structured and spent weeks in a lab dissecting strands of fibres and how the fat and blood is retained," says founder Simeon Van der Molen. Beetroot is included for juiciness and the pleasing, deep-red flavour it imparts when cooked (it was marketed as the first "bleeding" vegetable burger).

Although vegan diets are known to be healthier than meat-heavy ones, it seems unlikely that the new meat substitutes will advance that cause any further. An 85-gram (3-ounce) Impossible Burger will give you 220 calories, 13 grams of fat and just over a gram of salt. In terms of nutritional sins, that is no better than a typical beef patty. In fact, the Impossible Burger is actually heavier on the salt and saturated fat – as you might expect for something so processed.

But all of these products are substitutes for ground meat. Recreating the sensory experience of an intact cut, say a prime piece of rib-eye steak, is another story. That is where texture comes in. Flavour might seem like the most important quality to get right, but if a food feels wrong in your mouth, then the eating experience will be ruined. That is why for many in the artificial meat industry, getting the texture right is absolutely key to broadening the appeal.

To produce the authentic ground beef texture needed for meat-free burgers, many researchers use extrusion. But there are other ways of adding more complex textures to meat substitutes. Atze Jan van der Goot at Wageningen University in the Netherlands has been trying to crack this for years. His technique starts with the usual suspects: soy and gluten protein powders, to which food colouring is added to give them a more appealing hue.

This mixture is then pumped with water into a specialised piece of equipment called a Couette cell, consisting of two cylinders, one of which rotates inside the other under slight pressure. This exerts a shear force on the proteins that causes them to elongate into fibres and wrap around one another. "We can control the fibrousness," says van der Goot. "We hope that our technology will be able to mimic more types of meat than currently meat analogue products do."

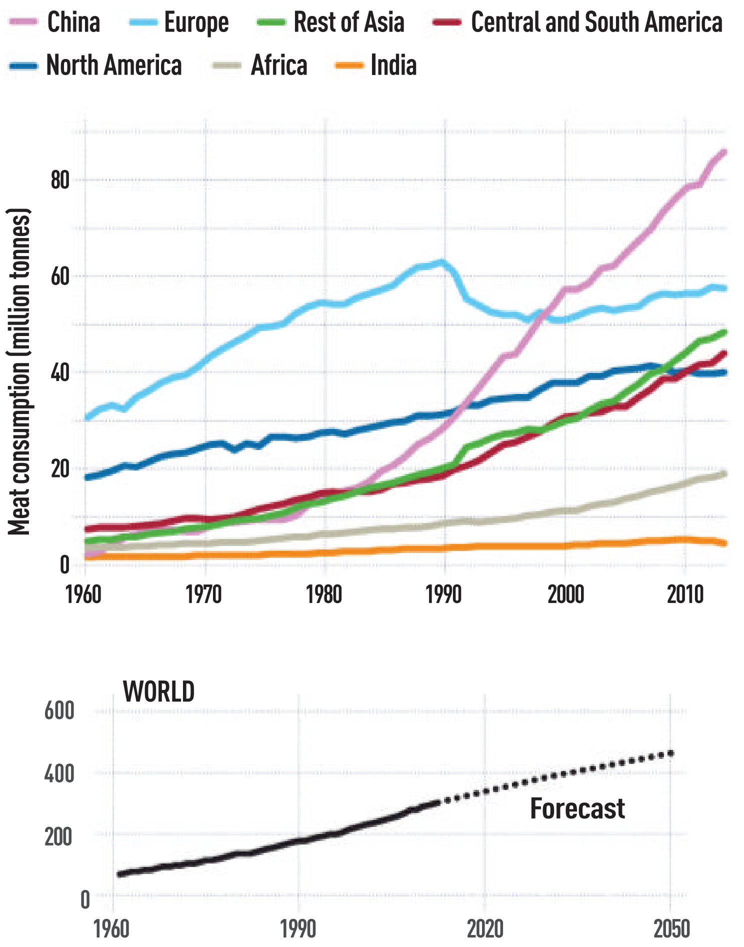
Not everyone is so gung-ho about Silicon Valley's latest attempt to save the world. Meat analogues have a place, but they aren't the whole story, says Tim Lang at the Centre for Food Policy at City, University of London. "I'm wary of technical fixes when we have a fundamental food system that is out of control," he says. "I think there is room for them, but I don't think they are any replacement for the need to tackle the social norms." ▮

←
Turn back to chapter 5 for more on sustainable eating

LAB-GROWN MEAT

Another way being explored to make meat more sustainable is just to cut out the animal. Meat cultured in the lab from stem cells will soon be on our plates – but it might not be what we all expect.

The consumption of meat is increasing globally and this trend is expected to continue, even if it is dropping off in a few regions. This means there might be quite an appetite for non-farmed alternatives



SOURCE: WORLD ECONOMIC FORUM

THE dream of growing meat in a lab instead of on a farm goes back 25 years. The first patents were issued in 1995. In the early 2000s, NASA funded research with the aim of finding new ways to make nutritious food for long-distance space travellers.

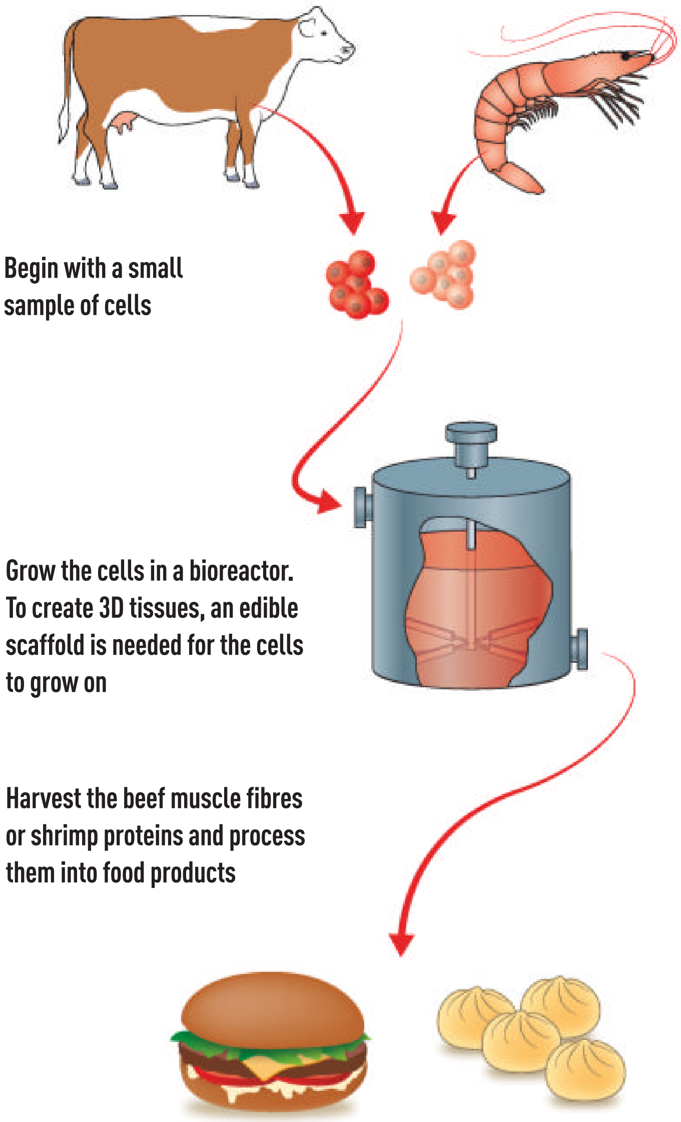
Things got more serious in 2013, when a patty made from cow muscle fibres grown in a lab was cooked and eaten at a press conference. Companies quickly sprang up all over the world, driven by a desire to right the wrongs of livestock farming. Unlike the real thing, cultured meat is almost cruelty-free: aside from biopsies to obtain stem cells, no animals are harmed. In theory, the environmental footprint – all that land, water and pollution – shrinks to almost nothing.

Back in 2013, the technology was nowhere near ready for the market; the burger took three months to grow at a cost of about €250,000. Mark Post at Maastricht University in the Netherlands, the scientist behind the project, said it would take 10 or 20 years to make it commercially viable. But things have moved faster than he anticipated. That doesn't mean cultured burgers, let alone steaks. Those are still at least five years away, according to Stephens.

Seafood, however, is a different story. Shrimp is only muscle and not any other tissue, so you don't have to worry about developing fat or connective tissue. "We might see the first commercial sale soon," says Stephens. Shrimp or other crustacean meat will be followed by salmon, tuna and white fish and then mammal and bird meats. Other animal products, such as milk, leather and wool, are also in development.

As this "cellular agriculture" industry develops, new battle lines are being drawn. Cultured meat will ›

Meat can be made in the lab from a few starter cells


Begin with a small sample of cells
Grow the cells in a bioreactor. To create 3D tissues, an edible scaffold is needed for the cells to grow on
Harvest the beef muscle fibres or shrimp proteins and process them into food products

be the biggest disruptive technology to hit the food industry since genetic modification. How will the conventional meat industry respond – embrace the new technology or fight it tooth and nail? "It's all to play for in this space," says Richard Parr at the Good Food Institute (GFI), a US non-profit organisation that promotes the development of alternatives to animal products.

There are around 30 firms refining cultured-meat technology around the world. The main ingredient is a culture of muscle cells (often with fat cells too) growing on a support structure called a scaffold, bathed in a liquid medium containing nutrients and growth factors. The medium stimulates the cells to proliferate, whereupon they spontaneously organise themselves into muscle tissue, aka meat.

There are still technical challenges to overcome, principally scaling up production and getting the taste and texture right. Yet these are widely seen as solvable in the near future. Nobody has yet achieved mass production, but some companies can already produce enough meat at an affordable-enough price to launch a product in a restaurant.

WILD TYPE

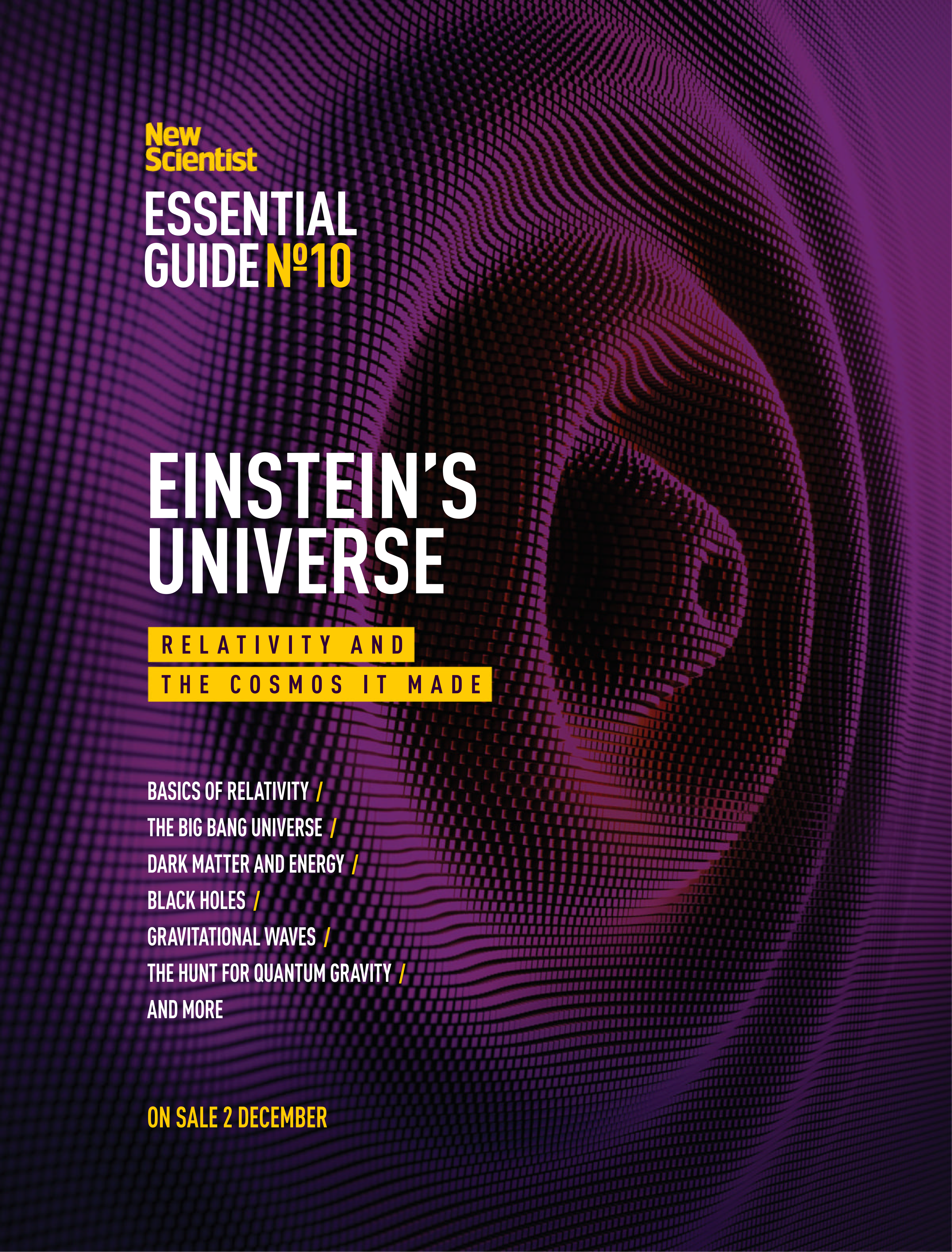
This cultured salmon was spawned in the lab, not the sea

The cleanness of cultured meat compared with farmed alternatives might be problematic. Conventional meat has a microbiome that – assuming the bacteria are benign – protects against food poisoning, because the resident bacteria outcompete hostile interlopers. But cultured meat comes out of the bioreactor sterile and is a sitting duck for bacteria. It may prove necessary to inoculate cultured meat with benign bacteria to eliminate this risk.

Another obstacle is red tape. Before cultured meat can be sold and eaten, regulators will have to be satisfied that it is fit for human consumption. As yet, it isn't clear how the regulatory system will work or what safety issues – if any – will come to light.

And even if the final product is excellent, that is no guarantee of acceptance. As proponents of genetic modification found, even demonstrably superior food can be doomed by consumer perceptions. In a recent survey of attitudes in the US, more than 60 per cent of people said they were willing to try cultured meat, but about 40 per cent said they were put off by its unnaturalness. What's more, until the technology matures, it is impossible to speculate whether it will be beneficial to the environment or not. Studies so far have provided conflicting evidence.

Full-scale launch into supermarkets is probably still several years away. Nonetheless, mass-market cultured meat is coming. The question, like so much with the future of food, is whether it will find a place on all our plates. ▮



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